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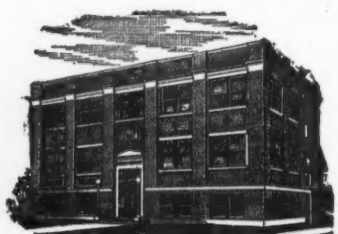

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MALIGNANT MYOMATA AND RELATED TUMORS OF THE UTERUS

REPORT OF 72 CASES OCCURRING IN A SERIES OF 4,000 OPERATIONS FOR UTERINE FIBROMYOMATA[1]

BY NEWTON EVANS, M.D., ROCHESTER, MINNESOTA

MALIGNANT tumors of the uterus comprise a numerically important portion of the malignant tumors in women. In the registration area of the United States in 1916 the malignant tumors of the uterus caused 23.4 per cent of all deaths from cancer in women (14). The larger number of the uterine cancers are, of course, epithelial cancers. Zacherl states that the proportion of non-epithelial malignant tumors of the uterus to carcinomata is 1 to 40 or 50. The records of the Mayo Clinic for the period of 1910 to 1918 show 22 border-line and malignant non-epithelial tumors of the uterus, while during the same period there were 873 cases of carcinoma of the uterus, a proportion of 1 to 40.

Notwithstanding this numerical disproportion there is in the aggregate a large number of malignant non-epithelial tumors encountered and reported. These, however, have received much less study than carcinomata of the uterus, and their pathology is poorly understood as compared with many other types of malignancy.

The literature on the subject is somewhat extensive, beginning with the writings of Virchow in 1860, and followed by papers by Ritter, 1887; Williams, 1894; Pick, 1895; Gessner, 1899; Weir, 1901; Jacobi and Wollstein, 1902; and others, who reviewed the principal features of the subject at the various periods. I shall not undertake to review this literature since excellent résumés have recently been written by Maroney, Geist, Proper and Simpson, and others. Kelly and Cullen in their book *Myomata of the Uterus* (1909) present a very interesting and informing discussion of the subject with a detailed description of a large series of cases.

Two questions with regard to the pathology of this group of tumors apparently have assumed great importance, namely: What is the histogenesis of malignant, non-epithelial tumors of the uterus? Do they originate in pre-existing fibroids? Most observers have concluded with regard to the first question that practically all spindle-cell tumors arising in the musculature of the uterus originate from smooth muscle cells and tend to differentiate into that type of cell. The most potent factors in helping to arrive at this conclusion have been the modern methods of studying and of differentiating the tissue cell types and, particularly, the application of the differential tissue stains of Mallory. The second question seems also to have been satisfactorily settled by the work of various observers who have demonstrated the existence of malignant myomata and cellular myomata within the structures of fibromyomata, other portions of which were of the

[1] Work done as a volunteer student in surgical pathology, under the direction of Dr. W. C. MacCarty, Mayo Foundation, April 16, 1919, to August 15, 1919.

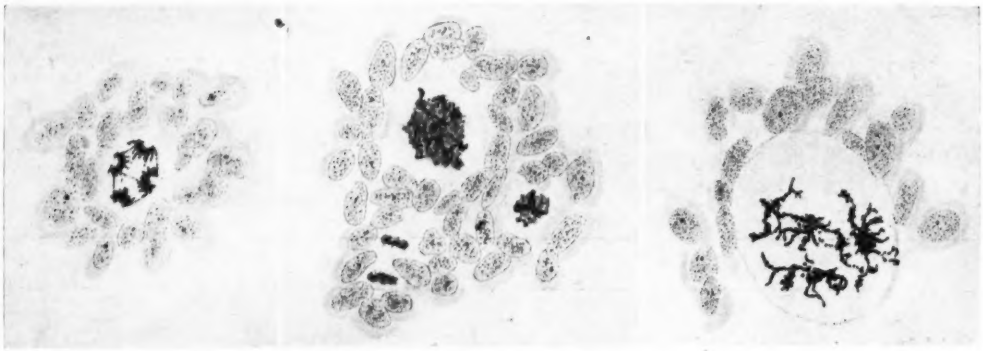


Fig. 1. Fig. 2. Fig. 3.

Figs. 1, 2, 3 and 4. Case 3, (125614). (Mitoses 7 600 in 1 cubic millimeter). Drawings of very large and atypical mitotic figures. Fig. 1 shows a very unusual form with four symmetrical rosettes.

ordinary benign tissue type. Williams, in 1894, gave this point careful study. The exhaustive work of Kelly and Cullen on their rather large series of cases also makes it plain that these tumors frequently originate within the structures of fibroids. The present observations lead to the conclusion that this insular transformation of otherwise benign fibroids is not usual, for it was not definitely evident in any of the large series of cases studied. It seems, therefore, that the tumor originates in the uterine musculature, and the structure remains unchanged, or that there is a gradual diffuse transformation in structure which leads to the formation found at the time of removal. This question in reality, however, is of academic interest only. The really important point is that these tumors appear as tumors, or within tumors which, clinically, and sometimes even at operation, cannot be distinguished from ordinary fibromyomata except in the very advanced stages of growth, when the extensive local infiltration and fixation of the tumor make it suspicious, or recognizable as definitely malignant.

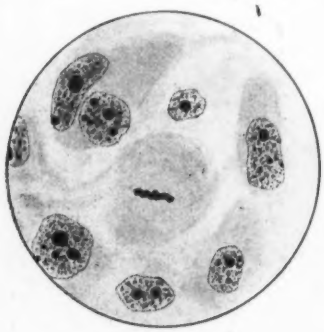


Fig 4.

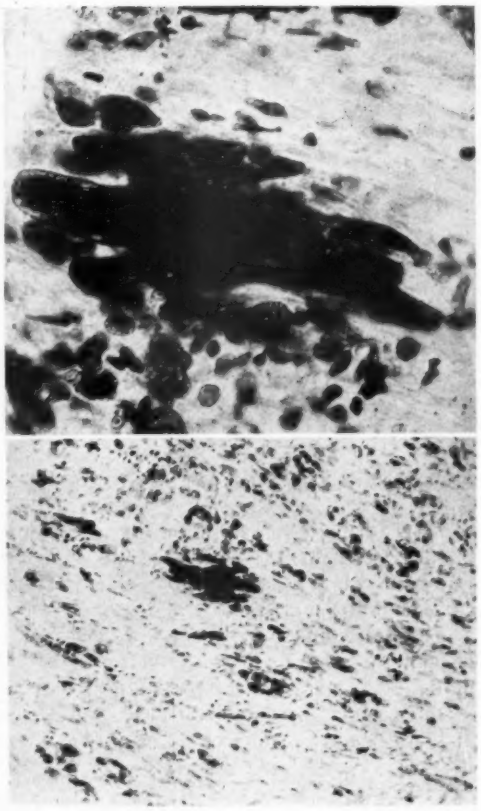
From a review of the literature in this field it is evident that there is a serious lack of understanding and agreement as to the histologic criteria which should govern the diagnosis of malignancy in this class of uterine tumors.

Kelly and Cullen found 17 cases among 1400 of uterine fibromyomata which they were willing definitely to denominate "sarcoma"; but in the same series there were 17 other tumors which they looked on as suggestive or suspicious of being sarcomatous.

Miller refers to the cases of Warnekross, who reported the surgical treatment of 7 cases of malignant myoma. Photographs of the tissues of these tumors were submitted to Aschoff who gave it as his unqualified opinion that in only 2 cases of the 7 could the tumors be classified as malignant.

In some of the numerous collected series of cases of uterine fibromyomata, the relative proportion which shows malignant change is given as low as 0.4 per cent; in others it is as high as 10 per cent.

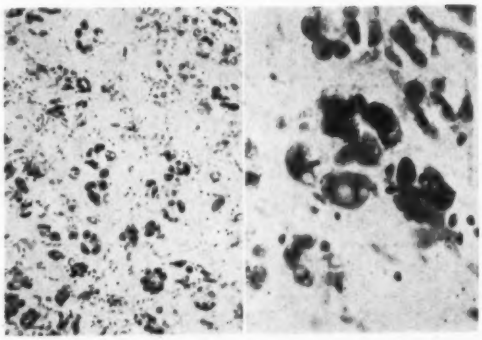
Maroney states that the diagnosis "must be a matter of individual interpretation in suspicious cases."



Figs. 5 (above) and 6. Case 29, (229935). (No mitoses). Photomicrograph high and low power, showing structure of a giant cell tumor, with one giant cell of immense size. Note the size of the immense irregular nucleus with some of the surrounding nuclei of tumor cells of ordinary size, and the relatively large amount of fibrous stroma. Tumors of this type did not recur after removal (x 100 and x 500).

In view of this evident lack of knowledge and lack of acceptance of uniform standards of malignancy as well as degrees of malignancy in this class of tumors, the principal purpose of this study is to make such comparisons of the histologic findings and the clinical histories of the material available as will serve to contribute something to the establishment of microscopic criteria.

A composite picture of these characteristics, as has been presented in the writings of several authorities, includes the following points:



Figs. 7 (at left) and 8. Case 33, (248318). (No mitoses.) Giant cell tumor, with very large nuclei arranged peripherally in the giant cells. Much fibrous stroma. Large nuclei contain vacuoles (x 100 and x 500).

1. Increase in size of tumor cells, as compared with normal muscle or benign muscle tumor cells.
2. Shorter and plumper cells with nuclei more nearly oval than normal muscle or benign muscle tumor cells, rounded, and "vesicular" nuclei.
3. Inequality in size and irregularity in shape and arrangement of the cells.
4. Lack of "differentiation" of cells.
5. Unequal staining of nuclei, and deeply staining nuclei.
6. Presence of immense cells (protoplasmic plaques) with hyperchromatic, single, or multiple nuclei (giant cells).
7. Presence of mitotic figures, typical and atypical.
8. Decrease or absence of stroma fibers between the cells.
9. Thinness or absence of vessel walls.

Kelly and Cullen in the descriptions of their 17 positive cases of malignant tumors appear, from a histologic standpoint, to place definite dependence on inequality in size and increase in the size of the tumor cells; and it is evident that they do not look on the presence of mitotic figures or of numerous mitoses as essential to the diagnosis of malignancy in these tumors. In 6 of the cases "nuclear figures" are noted, in 9 no mention is made of their presence, and in 2 it is stated specifically that they were not seen.

Ewing, in discussing the relative malignancy of the different malignant myomata,

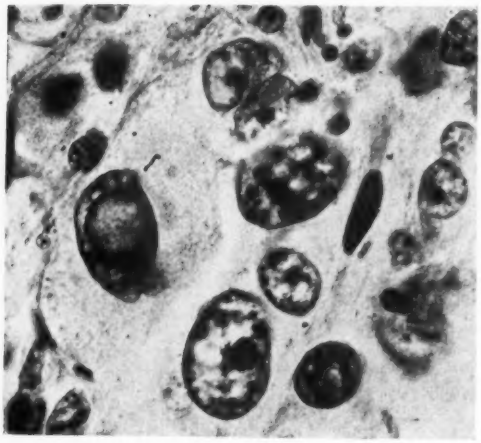


Fig. 9. Case 2, (77121) (Mitoses 2,400 in 1 cubic millimeter). Cells of immense size in a very malignant tumor. Very large dense nucleoli and nuclear vacuoles (x 500).

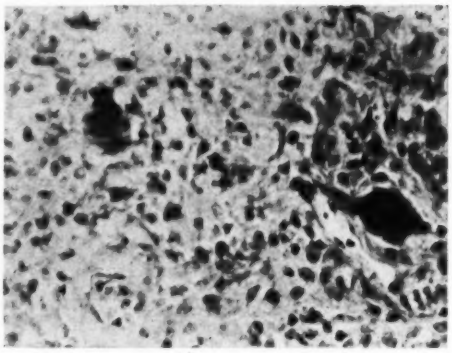


Fig. 10. Case 7, (64888) (Mitoses 2,560 in 1 cubic millimeter). Foreign body giant cells in a tumor with much fibrous stroma in its structure, but a highly malignant tumor (x 240).

says that the round-cell and the giant-cell structures are the most malignant.

Proper and Simpson, of the New York State Cancer Laboratory, in their recent article on "Malignant Leiomyomata," present an extremely valuable study of the microscopic characteristics of these tumors, since it is based on the correlation of the postoperative course of the patients with the microscopic structure of the tumors. They divide the tumors into three types varying in the degrees of malignancy which possibly represent stages of malignancy. With reference to their structure they state: "Histologically the tumors may vary from those made up of cells of uniform size resembling those which make up leiomyomata, but being somewhat shorter and plumper, with here and there a mitotic figure, up to tumors comprised of cells which are extremely irregular in size and shape, some being masses of protoplasm with giant or multiple nuclei, and showing all varieties of atypical mitotic figures." This statement seems to mean that the presence of the large irregular nuclei and giant cells is in direct proportion to the degree of malignancy and is an important accompaniment of the malignant process. This is in apparent accord with Ewing's statement. Proper and Simpson also make the very important observation that in doubtful cases they depend on the absence of mitotic figures as the criterion of a benign tumor. Mallory believes that the presence of mitotic figures in these myomata is a definite indication that they are capable of infiltration and are, therefore, malignant. Lockyer in his interesting volume on fibroids, recently published, says that malignant myomata are often difficult to distinguish and emphasizes the tendency to infiltrate and the presence of mitotic figures as a distinguishing characteristic.

The literature contains frequent accounts of metastasis by way of the blood stream of myomata which are said to have the structure of benign or ordinary fibromyomata. Lockyer refers to cases of this kind. Ewing states that, so far as he has been able to learn, no case has been fully studied in which definite variations from the usual structure were wanting, although in several instances these variations were not very pronounced. Strong is quoted by Maroney as making the very radical state ment relative to the existence of microscopic criteria for malignancy in these tumors, that the "only criterion is infiltration and destructive growth. Mere richness in cells, mitoses, and even irregularities in size of cell do not constitute sarcoma." Strong further states: "There never can be any absolute criterion for their malignancy and their interpretation will always be effected by the personal equation of the individual observer." Ewing, shar-

Fig. 11. Drawing of nuclei of tumor cells showing changes evidently those of direct nuclear division-amitosis. Series showing progressive changes in long, oval nuclei of ordinary size.

ing the uncertainty of practically all observers relative to the microscopic diagnosis, says: "Sarcomatous tendencies and precancerous changes do not constitute real sarcoma or cancer." It is thus evident that many "very cellular" tumors or myomata exist which give difficulty in classification as far as their real malignancy is concerned and which are sometimes classified by the pathologist as malignant, and sometimes as benign or doubtful.

Mitotic figures. From the foregoing statements and other observations it may be assumed that, in a general way, the presence in the tumor tissue of evidences of indirect cell division is looked on as a characteristic of the malignant growths of the types under consideration. So far as I can learn, however, no attention has been given to a determination in a given case, of the actual or relative numbers of mitotic figures as related to the degree of malignancy of the tumor, or whether or not any such relation exists. The facts presented in the present series of cases show that such relationship undoubtedly does exist.

This series comprises 72 cases diagnosed as "sarcomatous," "cellular," or "very cellular" fibromyomatous tumors in the Laboratory of Surgical Pathology of the Mayo Clinic in the years from 1906 to 1918 inclusive. All tumors of this kind observed in about 4,000 operations for the cure of uterine fibromyomata are included.

Table I serves to show the very definite relationship between the more evident characteristics of the cellular structure of the tumors, particularly the presence and relative proportions of the mitotic figures, and the clinical outcome of the disease. In each case

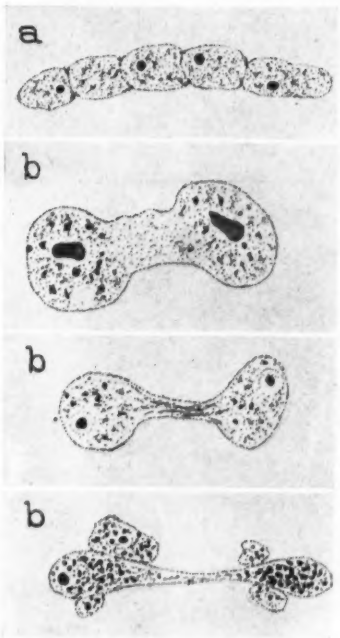


Fig. 12. Drawing *a*. Result of multiple change of a long nucleus. *b*. Forms seen in giant cells of tumors having no mitotic figures.

evidences of indirect cell division were searched for and careful estimates were made of the actual number of these figures in a given area of the tumor tissue; the numerical values were expressed as the number of mitotic figures seen in 100 microscopic fields of a 1/12th oil immersion lens. These values are also translated in an adjoining column into the number of dividing cells in a cubic millimeter of the tissue, taking into consideration in this estimation the thickness of the tissue section.

The tumors readily divide themselves into three definitely delimited groups, on the basis of the number of mitotic figures present. In the first 13 cases the tumors show from 2200 to 12000 mitotic figures for each cubic millimeter. Cases 15 to 25 form a group having from 200 to 800 in a cubic millimeter. In the remainder of the cases the tumors either contained no figures on examination or only a very few, one or two being found after long

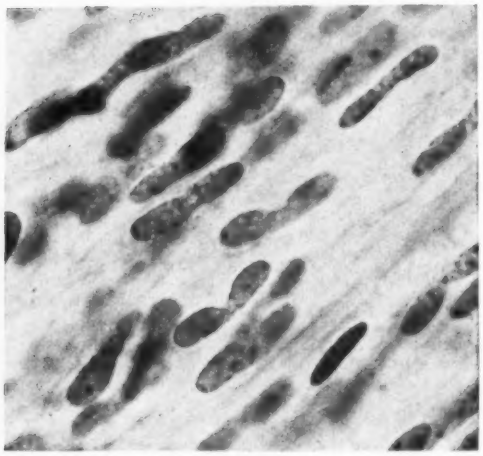


Fig. 13. Case 61, (248302). (No mitoses). Photomicrograph showing numerous long oval nuclei in direct division (x 1000).

searching. One of the most noticeable features of the tabulation is the definite numerical separation between the first and the second groups, no tumors being found which showed a numerical value between 800 and 2000. It would not be justifiable to conclude that in a larger series of tumors or in another similar series, values within this hiatus might not be found, yet the numerical distinction is so clear-cut that it cannot escape notice.

The real importance of such distinctions, however, becomes evident when the after-course of these cases is considered. Of the 13 patients in the first group, 11 had recurrences within periods of from 1 month to 18 months. Only 2 patients are known to be living; they give no indication of a return of the malignant tumor, 7 and 4 months respectively, since operation, periods too short to preclude the possibility of later recurrence. However, since the recurrences were rapid in the other cases, one is justified in hopeful prognoses in these 2 cases.

In definite contrast to the cases in the first group, in the 11 cases in the second group, having mitotic figures in the proportion of 200 to 800 to each cubic millimeter, there has been no mortality; at least in the cases about which we have been able to obtain a recent report, 9 of the 11, and there are good reasons for believing that the same is true in the other 2 cases.

In the remaining group of 48 cases, in which no mitotic figures are seen, or only a very few, there is no mortality from recurrence, at least in a large majority concerning which it has been possible to obtain recent reports.

This striking evidence of the importance of numerous mitotic figures as an indication of definite malignancy makes it desirable to study the reports of similar series of cases from this viewpoint. The very complete description of the histology and the clinical features of Kelly and Cullen's 17 cases makes an interesting comparison possible. Eleven of their 17 patients survived the operation and so are available for comparison on the basis of after-history. Only 4 of the 11 died of recurrence. In each of these 4, mitotic figures are mentioned as being present, in one "many" were seen, and in one other as many as "6 in a field." Of the remaining 7 patients without recurrence, only one tumor is mentioned as showing mitotic figures, and in this there was said to be "one here and there." One might be justified from a study of these descriptions in the opinion that the 4 fatal recurring tumors belonged in the same group with those of this series having many mitotic figures, and that the one tumor which had mitotic figures "here and there" belonged in the next group having fewer mitoses and clinically showing no recurrences. It might be questioned whether Kelly and Cullen are entirely right in classifying in their group of 17 definitely malignant "myosarcomata" the 9 cases which are not mentioned as showing mitoses, and 2 which are described specifically as not showing such figures. In none of these patients having tumors with no mitoses recorded, who survived operation, was there any recurrence.

A personal communication from Dr. Simpson relative to the series of 22 cases of this type of tumor recently reported by Proper and Simpson indicates that the findings of their specimens, with reference to the relative number of mitotic figures in sections of the tumors of their different histologic types, are substantially in harmony with my series.

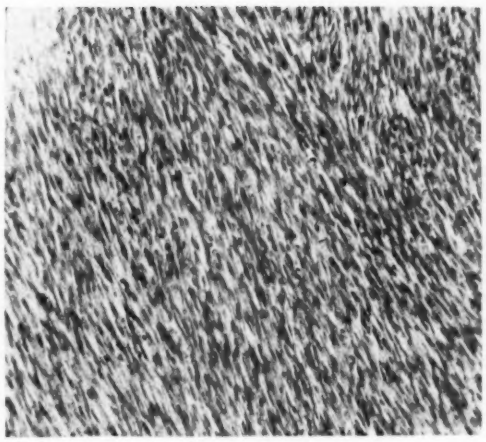


Fig. 14. Case 8, (38352) (Mitoses 4,000 in 1 cubic millimeter). Marked degree of polarity and alignment of the most malignant tumor as compared with the irregular arrangement of the cells in the less malignant tumor.

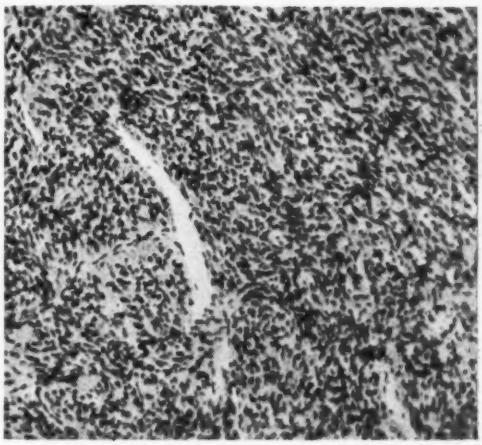


Fig. 15. Case 27, (215251). (No mitoses). See Figure 14.

While we are considering the question of mitosis it would be well to mention the frequent occurrence, in the tumors composed of a mass of very large sized tumor cells, of numerous mitotic figures of unusual form and gigantic size. The drawings in Figure 1 show several examples, including the unusual figure of a double division resulting in the formation of four newly formed symmetrical rosettes. These large and so-called atypical mitotic figures are unquestionably always a sign of high grade malignancy.

Giant cells and hyperchromatic nuclei. Another histologic feature which demands consideration in a study of these tumors is the presence in a certain proportion of them, of cells with large, irregular, hyperchromatic, and usually multiple nuclei. Some of these are so immense as almost to exceed belief (Figs. 2, 3, and 4). The importance of these changes in the diagnoses of malignancy or degree of malignancy, judging from the statements in the literature of the subject, is usually considered to be very great. A study of Table I will give a basis for rather definite conclusions in regard to this point.

It is true that in the first group of 13 cases, including all of those of the series in which there was postoperative recurrence, giant cells were present in varying degree, but in a considerable proportion of them in comparatively small numbers. Most of the tumors in the next group of the series (Cases 14 to 24), none of which recurred, tumors which were characterized by a relatively small number of mitotic figures, had no giant cells, and only a few had any giant cells. The phenomenon of greatest interest, however, is that in many of the tumors of the third group (Cases 25 to 72) giant cells were numerous and in a few tumors they were present in such numbers, and of such size and complexity as to attract attention. It should be remembered that in this group there were no recurrences and histologically the tumors were characterized by the absence of mitotic figures or by the presence of only a very few at the most. A review of the literature indicates that such tumors have usually been looked on as malignant. The facts evident in this series compel the conclusion that there is nothing to indicate that the presence of the giant cells alone is an indication of a high degree of malignancy or indeed of malignancy at all. In the tumors showing the greatest tendency to giant cell formation but without mitotic figures, it is invariably found that the cells are not closely packed but are separated by large amounts of fibrous stroma with a tendency to hyalinization. Kelly and Cullen believe that the frequent association of this

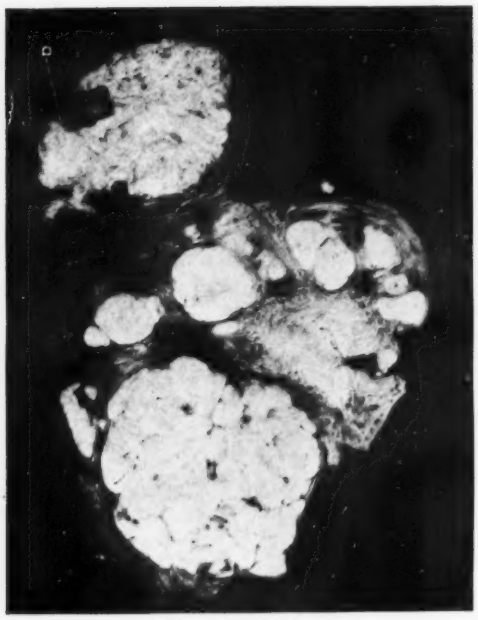


Fig. 16. Case 43, (76296) (Very few mitoses). Gross appearance of section through a fixed specimen of uterus containing multiple fibromyomata. One of the tumors, *a*, is very cellular. Its yellow color is indicated by the darker shade in the photograph. It contains an area of hæmorrhage.

type of cell and stroma indicates malignancy and that the presence of the hyalinization is an important factor in its pathogenesis. It may be concluded that these cellular changes are a phenomenon of degeneration coincident with fibrosis and hyalinization rather than a manifestation of the exaggerated productive and reactive phenomena which characterize real malignant tumors. The origin of the cells which take on this unusual type of change is somewhat uncertain but from the cellular relations it seems that many of these very large mononucleated and multinucleated cells are modified muscle cells while others, from their position in relation to minute vascular channels, appear to originate from endothelial cells.

The morphology of these giant cells is distinctly different from that of the so called foreign body giant cells which are found in many tumors, in the lesions of the infectious granulomata, and in the vicinity of various

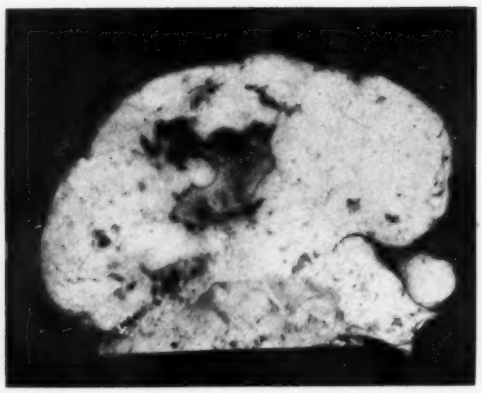


Fig. 17. Case 11, (213999) (Mitoses 12,000 in 1 cubic millimeter.) Natural size of a section through the fixed specimen. The smooth appearance of the cut surface is evident in contrast to the fibrous appearance of the ordinary fibroid.

foreign bodies in the tissues. Those appearing in the tumors in our series should be classed as true tumor giant cells. In a few of the tumors studied, however, there is a tendency to the formation of the foreign body giant cells. This tendency is marked in only one of the tumors (Case 7, Fig 5). In these figures numbers of giant cells of this type are scattered throughout various portions. They are characterized by a compact, densely acidophilic staining cytoplasm with numerous small oval or round nuclei centrally located. These cells probably have their origin in modified connective tissue stroma cells or in some type of wandering cell. In contrast with these cells are the characteristic tumor giant cells which have irregularly shaped, lightly staining, indistinct bodies with very large, unequally sized, irregularly shaped, deeply staining nuclei usually arranged in a ring at the periphery of the cell body. Many of these nuclei have very large densely staining chromatin granules, and often the chromatin is disposed in threads (Fig. 2). In many of the sections of the very malignant tumors many very large cells are found undergoing unusual forms of mitosis. But typical multinucleated giant cells with nuclei in the stages of mitosis have not been found. This leads to the conclusion that the true tumor, multinucleated giant cell forms by the direct, or

amitotic division of the nucleus. Such dividing nuclei are in fact frequently seen.

Direct cell-division of tumor cells. Throughout the tissue of a large proportion of these uterine tumors are seen nuclear forms, the morphology of which can be interpreted only as that of cell division by the direct or amitotic method. This is particularly true of those tumors which do not belong to the more malignant type, containing no mitotic figures, and the growth of which cannot be accounted for by mitotic cell division. The drawings and the photographs of nuclei thus dividing will give a general impression of the phenomena as seen in the sections. There are two distinct forms of such nuclear division. One of these is seen in the large nuclei of the large hyperchromatic giant cells described in the preceding paragraph. These nuclei may divide equally, but more often they divide unequally by a process of lengthening and constriction at the middle; the two portions are pulled apart, a large strand of nuclear material connecting them, thus assuming a distinct dumb-bell shape (Fig. 6). The other form of direct nuclear division is seen in the tumor cells of ordinary size, of long or short oval shape, or with rounded nuclei. The cleavage is usually preceded by a distinct indentation on one side of the nucleus before it is separated into two equal parts. The line of cleavage is in a distinctly oblique direction, the degree of obliquity being greater in the shorter and plumper nuclei (Fig. 7). Occasionally nuclei of great length will be seen evidently dividing almost simultaneously by a multiple cleavage so that a chain of attached oval nuclei results (Fig. 6).

The conclusion is apparently justified that the direct cellular division observed accounts for the tumor growth in the growing tumors which show no signs of growth by mitosis. The subject of amitotic cell division is discussed at some length by Wilson in his book on the cell. He emphasizes three points of special interest in this connection:

1. Cells undergoing amitotic division have a tendency to become larger in size than other cells of the same tissue type.

2. The cells have a tendency to nuclear division without division of the cytoplasm, thus producing multinucleated giant cells. Both of these tendencies are very definitely illustrated in the large nuclei of the tumor giant cells which so frequently characterize these tumors and especially those not showing definitely malignant tendencies.

3. Direct cell division is an indication of degeneracy of the cells involved.

On this last point Wilson quotes von Rath: "When once a cell has undergone amitotic division it has received its death warrant; it may indeed continue for a time to divide by amitosis, but invariably perishes in the end." Wilson states, however, that this is probably an extreme view as there are definite examples to the contrary in lower forms of life. It may be assumed, therefore, that the direct nuclear division which was so frequently seen in the tumors of this series undergoing fibrosis and hyalinization as well as the marked tendency to giant cell formation under the same conditions is a manifestation of biologically regressive cellular changes.

Kimura has recently reported an interesting and important piece of work which throws additional light on the problem of the relation of mitotic figures (or evidence of frequent indirect cell division) to malignancy. The purpose of his experiments is to show the influence of the X-ray on the growth and invasive power of malignant tumor tissue, using susceptible animals (mice) and artificial tissue cultures in parallel series. An appropriate dosage of X-ray rendered the cancer tissue incapable of invading the susceptible animals but the artificial cultures of the irradiated cancer tissue grew just as freely as the control tissue cultures. The remarkable fact was noticed that the tissue exposed to the X-ray grew in artificial culture without any mitotic figures while the control tissue grew with large numbers of mitotic figures. The type of cell division concerned in the growth of the tissue which had lost its invasive power in animals must have been the direct or amitotic type. It seems that the process of mitotic cell division had some relation to the malignancy of the tumors and that with the loss of their mitotic figures they lost their invasive powers. These interesting results are in harmony with the facts observed

TABLE I.—TABULATED FINDINGS IN 72 CASES OF MALIGNANT MYOMATA AND RELATED TUMORS OF THE UTERUS

	Case	Mitotic Figures[1]		Cells	Giant cells	Operative death	Recurrence	Death after operation	Diagnosis at operation
		In 1/12 microscopic field	In cmm.	Size and shape					
1	33870	120/100	4800	2, spindle	Many	No	Yes	4½ mos.	Sarcoma
2	77121	60/100	2400	4, round, oval and mixed	Many	No	Yes	?	Sarcomatous
3	125614	190/100	7600	3, spindle and mixed	Few	No	Yes	9 mos.	Sarcoma?
4	207128	300/100	12000	3, mixed	Many	No	Yes	6 wks.	Spindle-celled sarcoma
5	239225	175/100	7000	2, round, oval	Many	No	Yes	12 mos.	Mixed-celled sarcoma
6	55911	55/100	2200	2, spindle	None	No	No, 7 mos.	No	Sarcomatous
7	64888	64/100	2560	2, spindle, round	Foreign body	No	Yes	3 mos.	Degeneration and necrobiosis[2]
8	38352	100/100	4000	2, oval and mixed	Few	No	Yes	6 wks.	Sarcoma
9	111435	93/100	3720	3, round, oval and mixed	Few	No	Yes	1 mo.	Sarcoma
10	119219	141/100	5640	1, spindle, oval	Few	No	Yes	17 mos.	Necrobiosis[2]
11	213833	58/100	2320	2, oval	Few	No	Yes	2 mos.	Sarcoma
12	213999	300/100	12000	2, round, oval	Few	No	Yes	13 mos.	Sarcoma
13	255090	120/100	4800	1, mixed	Few	No	No, 4 mos.	No	Sarcoma
14	45651	14/100	560	2, round, oval	None	No	No, 9 yrs.		Degeneration. Fibroid
15	85077	5/100	200	2, round, oval	None	No	No, 3 yrs.		Myxosarcoma. Degeneration
16	103207	13/100	520	1, oval	None	No	No, 5 mos.		Sarcomatous fibroid
17	169459	10/100	400	2, round, oval	None	No	No, 3 yrs.		Angiomatous fibroid
18	176816	20/100	800	1, round, oval	None	No	No, 1 yr.		Sarcoma[4]
19	184003	17/100	680	2, round, oval	None	No			Sarcoma
20	186703	5/100	200	1, spindle, oval	None	No	No, 2½ yrs.		Early myxosarcoma
21	201753	6/100	240	1, oval	None	No	No, 10 mos.		Spindle-celled sarcoma
22	224262	6/100	240	1, spindle, oval	None	No			Myosarcoma
23	239181	20/100	800	2, mixed	Few	No	No, 1 yr.		Myosarcoma
24	1591	8/100	320	Varies	Many	No	No, 13 yrs.		Sarcoma
25	93251	None	None	1, spindle, oval	Few	No	No, 3½ yrs.		Sarcoma?
26	187053	Very few	....	2, mixed	Few	No			Secondary hypertrophy
27	215251	None	None	1, mixed	Small areas	No	No, 1½ yrs.		Cellular
28	224819	Very few	...	1, spindle, oval	Few	No	No, 16 mos.		Fairly cellular
29	229035	None	None	Varies	Many	No	No, 14 mos.		Sarcomatous
30	238918	None	None	2, mixed	Few	No	No, 1 yr.		Myosarcoma
31	241253	None	None	2, spindle, oval	Few	No	No, 11 mos.		Fairly cellular
32	242747	None	None	2, mixed	Few	No	No, 11 mos.		Secondary hypertrophy
33	248318	None	None	2, mixed	Many	No	No, 10 mos.		Malignancy
34	265765	None	None	2, round, oval	Small areas	No	No, 3 mos.		Fairly cellular
35	6613	Very few	....	1, spindle, oval	Few	No			Sarcoma
36	9105	Very few	....	2, spindle, oval	Few	No	No, 11 yrs.		Sarcoma
37	37931	None	None	1, spindle, oval	Few	No	No, 9 yrs.		Sarcomatous degeneration
38	51308	None	None	1, spindle, oval	None	No	No, 8 yrs.		Sarcoma?
39	59317	None	None	1, mixed	None	No	No, 5 yrs.		Sarcomatous changes?
40	59792	None	None	1, mixed	None	No			Degenerative sarcoma?
41	54214	None	None	2, mixed	None	No	No, 7 yrs.		Sarcomatous degeneration
42	75009	None	None	1, mixed	None	No	No, 5 yrs.		Fibrous sarcoma or myosarcoma
43	76296	Very few	....	2, spindle and oval	None	No			Sarcomatous
44	88314	Very few	....	1, mixed	None	No	No, 6 yrs.		Sarcoma
45	94437	Very few	....	1, spindle, oval	None	No	No, 18 mos.		Sarcoma?
46	132034	None	None	2, mixed	None	No	No, 5 yrs.		Cystic degeneration
47	170677	None	None	2, spindle, oval	None	No	No, 2 yrs.		Hæmangioma
48	180101	Very few	....	1, spindle, oval	Few	No			Secondary hyperplasia
49	136805	None	None	2, spindle, oval	Few	No	No, 2½ yrs.		Secondary hyperplasia.
50	189824	Very few	....	2, round and oval	None	No	No, 2 yrs.		Very cellular
51	194810	None	None	2, spindle, oval	None	No			Fairly cellular
52	197431	Very few	....	2, spindle, oval	None	No	No, 2 yrs.		Secondary hyperplasia
53	207489	None	None	1, spindle, oval	None	No	No, 9 mos.		Early myosarcoma
54	208063	None	None	1, spindle	None	No		Died 3½ mos.	Celular
55	220349	Very few	...	1, mixed	None	No			Cellular
56	224532	Very few	....	2, mixed	None	No	Metastasis?		Early sarcoma
57	228019	Very few	....	2, mixed	None	No			Fairly cellular
58	228853	Very few	...	1, irregular	None	No	No, 15 mos.	No	Secondary hyperplasia
59	237287	None	None	1, spindle, oval	None	No			Cellular areas, secondary hyperplasia
60	243528	None	None	1, irregular	None	No	No, 1 yr.	No	Very cellular
61	248302	None	None	2, spindle, oval	Few	No	No, 7¼ mos.	No	Cellular
62	254173	None	None	1, spindle	None	No	No, 7 mos.	No	Myosarcoma[5]
63	258667	None	None	1, spindle, oval	Few	No	No, 5 mos.	No	Cellular, probably early sarcoma
64	258698	Very few		1, round, spindle	None	No	No, 5 mos.	No	Spindle cell sarcoma
65	258852	None	None	1, irregular	None	No	No, 5 mos.	No	Fairly cellular
66	259958	None	None	2, irregular	None	No		No	Early sarcoma
67	175048	No specimen				No			Fairly cellular
68	199649	No specimen				No			Fairly cellular
69	205331	No specimen				No			Fairly cellular
70	247494	No specimen				No	9 mos. ?		Cellular, degenerating
71	250107	No specimen				No	No, 9 mos.		Very cellular
72	207005	No specimen				No	Inoperable	14 mos.	No examination

(For footnotes see opposite page.)

in the group of malignant uterine tumors in this series since only those tumors which contained large numbers of mitotic figures were able so successfully to invade the tissues as to recur after removal.

Tumor cell differentiation. While it is true that in many of the more malignant of these tumors there is a decided decrease in those characteristics which are considered as signs of differentiation, still in practically all a greater or lesser degree of spindle shape, polarity, and alignment of the tumor cells is recognizable. In some of the most malignant this is very pronounced, much more so than in many of the cellular tumors which evidently have little if any real malignancy. This may be easily seen by comparing the appearance of the sections in Figure 8. Therefore, it is evident that the presence or absence of these characteristics is not an essential criterion.

The one dependable microscopic feature so far considered is clearly that of abundant mitotic figures in the extremely malignant cases. The question of the existence of other constant criteria should be considered. It seems to me that the most important microscopic features aside from the frequency of mitoses are:

1. The large size of the great mass of the tumor cells in a given case, and a marked inequality in their size. In those very malignant tumors which are characterized by this type of cellular structure many abnormally large mitotic figures are apparently invariably found (Fig. 1), and such figures otherwise unusual in their morphology.

2. The relative decrease in the amount of fibrous stroma.

3. The growth among the tumor cells of blood vessels with very thin walls or with walls entirely wanting.

4. The relative increase in the size of the nucleus of the tumor cells as compared with the mass of the cytoplasm of the cell body.

While all these changes are important, careful study of the material shows that none is constant, that is, invariably present in the very malignant tumors and always absent in those not malignant. Notwithstanding this fact it is well to note that the tumors composed of a mass of closely packed cells of very large size are invariably extremely malignant, although it is not true that all the malignant tumors have this particular morphology.

It would be rash to maintain that other constant, easily recognizable microscopic criteria of this particular type of malignancy do not exist; but up to the present time I have not been able to recognize any single criterion which is constant aside from the peculiarities in mitoses, although the presence of several of the prominent characteristics when occurring in combination may possibly be sufficient to insure the accuracy of the diagnosis of malignancy without giving any attention to the question of mitotic figures.

Frequency of occurrence. Reference to the literature indicates that frequently efforts have been made to estimate the incidence of the malignant non-epithelial tumors of the uterus as compared with the total number of cases of uterine fibroids. Table II gives an idea of some of the results. The wide variation in these estimates is no doubt largely due to the absence of uniform standards rather than to actual variation in the incidence of the malignant tumors.

The 72 cases in the present series in which the cellular and definitely malignant tumors are included were examined in the Mayo Clinic during the years 1906 to 1918 and 3 months of the year 1919, during which time operation was done in approximately 4,000 cases of uterine fibroids. However, in order

FOOTNOTES FOR TABLE I

[1] The computation to determine the number of mitotic figures in a cubic millimeter of tumor tissue: Diameter of oil immersion field—0.170 mm.; radius—0.085 mm.; area of one field—(3.1416 times the square of the radius) 0.023698 sq. mm. Taking the average thickness of section as 0.011 mm. the cubic contents of one microscopic field would be 0.000249678 cmm. or approximately 0.00025 cmm., or 1/4000 of a cmm. Therefore having determined the number of mitotic figures in 100 fields the number in a cubic millimeter would be 4000 times the average number in one field or 40 times the number in 100 fields.

[2] A vaginal myomectomy. At first operation the malignancy was not discovered, but it was discovered at a second operation following recurrence.

[3] At first operation, a subtotal abdominal hysterectomy was done; the malignancy was not recognized. A second operation was done because of recurrence.

[4] In this case the removal of the tumor mass was not accomplished, but was discontinued before completion. A tube was left in the abdominal wound to facilitate radium treatments, of which the patient subsequently had a number; also X-ray treatments were given. The patient was alive at the end of a year.

[5] The patient died three and one-half months after the hysterectomy from an acute peritonitis, following a cholecystectomy; the death had no relation to the previous conditions.

[6] Fragments only removed. Tumor regarded as inoperable and not removed.

to obtain relative figures which will have much value as an indication of the real incidence of these tumors of varying degrees of malignancy, it will be necessary to make the comparison from material collected over somewhat shorter periods, since only during the past few years has sufficiently painstaking investigation been made of all the fibroids which are removed at operation to insure the recognition of practically all the unusual myomata, particularly those of the lesser degrees of malignancy. Among the 968 cases of operation for removal of fibromyomata during the two years 1917 and 1918, were 6 cases of the most malignant type, those tumors containing from 2,200 to 12,000 mitotic figures to the cubic millimeter. When the tumors having from 200 to 800 mitotic figures to the cubic millimeter are included there are 12 in all; and including all these with the remainder of the series, that is, those having the cellular structure but lacking the frequent mitoses, gives 38 in all. The percentage for the first group was 0.62, for the first and second groups together 1.25, for the three groups 4.00.

During the period from 1910 to 1918 inclusive the total number of fibroid operations was 3,297; only 13 cases (0.39 per cent) belonged to the very malignant group; adding the 9 cases of the second group, there are 22 cases (0.67 per cent). The figures for the third group of this longer period are too inexact to be included. When the figures for the two year period and those for the nine year period are compared the percentages for the shorter period are found to be definitely larger; and it is probably safe to conclude that these larger percentages more nearly represent true conditions.

A comparison of the percentages for the two year period with the figures taken from those of other observers, as shown in Table II, leads one to believe that in a rough way those series which give percentages less than 1 per cent are made up of only the most malignant tumors, those in Group 1. This would include such groups of cases as Broun's 1,500 cases at the Woman's Hospital, New York, and the 337 cases of Noble. Those series giving percentages between 1 and 2 are probably made up largely of cases that would be included in our Groups 1 and 2. The higher figures are no doubt in series which include not only the more malignant types but also practically all of the cellular tumors, the majority of which manifestly show little malignancy clinically and, as we have shown, contain few or no mitotic figures.

TABLE II.—FREQUENCY OF NON-EPITHELIAL MALIGNANT UTERINE TUMORS AS COMPARED WITH FREQUENCY OF FIBROIDS

	Fibroids	Malignant	Percentage
Proper and Simpson	357	22	6.00
Miller	9750		1.96
Lewis	1518		1.40
Olshausen	6470		1.30
Kelly and Noble	2274		2.00
Winter	500		3.20
Winter	253		4.30
Noble	337	2	0.60
Kelly and Cullen	1400	17	1.20
Martin	205	4	1.90
Fehling	409	8	1.90
Broun, Woman's Hosp., N. Y.	1500	7	0.40
Warner	100		
Including all "cellular"		7	7.00
"Sarcomata" only		2	2.00
Geist	250	12	4.80
Present series	968	Group 1, 6	0.62
1917–1918		Groups 1 and 2, 12	1.25
		Groups 1, 2 and 3, 38	4.00
1906–1918	3297	Group 1, 13	0.39
		Groups 1 and 2, 22	0.67

Degrees of malignancy and relation of the different types of tumors. Three questions still remain to be considered. (1) The degree of malignancy of the tumors which are classified outside of the group of manifestly malignant tumors, but including tumors none of which recurred after removal (Groups 2 and 3). (2) The question of the biologic relationship between these less malignant tumors and the most malignant ones. Are they a fixed type of tumor, or are they simply in a stage of metamorphosis, representing a transition stage between the ordinary fibroid with its mature, fully differentiated type of cell and the real cancer? (3) Is it not possible that these very cellular myomata with short spindle cells and short plump nuclei and an occasional mitotic figure are simply ordinary fibromyomata in an actively growing phase, and at a later period may they not cease their active growth and become ordinary fibroids with the structure that the majority of fibroids possess? With regard to the last of these questions it seems possible that there may be a stage of growth in which the balance may turn in either direction, on the one hand,

back to the fully differentiated type and, on the other, to a still more active tumor growth in which the cells vary more widely from the adult type and in which the increased rate of growth and increased power to invade tissue is indicated by an increase in mitotic figures.

With regard to the first two of these questions the positive opinion seems justifiable, although not proved, that the actively growing tumors which contain an appreciable number of mitotic figures are in a stage of transition toward actual malignancy and, if undisturbed, will become malignant.

A tentative classification of the tumors of this series, exclusive of those in the first group, those definitely malignant, is as follows: Group 2 includes those with a mitotic figure, content of from 200 to 800 to the cubic millimeter and should be looked on as in a transition stage, border-line tumors between the definite malignant group and the remaining group of cellular tumors. The cases in Group 3 are premalignant and presumably have malignant tendencies.

Gross characteristics. These uterine tumors appearing in the locations of, and in form and general appearance resembling fibromyomata, have a color and consistency which as a rule are characteristic; and it should be remembered that they occur frequently in the uterine ligaments and other locations where fibromyomata are found. The color is difficult to describe but is remembered when once seen and recognized. It may be said to be a shade including pink, yellow, and gray. Fixed gross specimens have a yellowish tinge, which distinguishes them from the ordinary fibroid. The tissue is much softer and has a smooth homogenous cut surface as compared with the firm fibrous surface of the usual fibroid, and is decidedly more friable. In the definitely malignant forms the tumor mass is still more friable and varies in color, due to hæmorrhage and degenerative and necrotic changes. In most of the tumors of the definitely malignant type, the infiltration and destruction of the uterine and other pelvic structures involved is evident, but the less malignant forms are usually as definitely delimited from the surrounding myometrium as is the ordinary fibroid.

TABLE III.—MORTALITY PERCENTAGES FOR CONSECUTIVE OPERATIONS FOR REMOVAL OF FIBROIDS OF THE UTERUS

	Cases	Mortality Percentage
Woman's Hospital, New York (Broun)	1500	1.86
Woman's Hospital, New York (1918)	262	1.53
Deaver	750	1.73
Frank and Brethauer	400	1.75
St. Mary's Hospital (Mayo Clinic, 1916, 1917, and 1918)	1188	1.18
St. Mary's Hospital, (1912–1918)	2774	1.50

Metastasis. Definite indications of metastasis to distant organs were not found in any of these cases. From the findings at operation and from the subsequent histories of the fatal cases there was evidence of extensive local and abdominal metastasis.

Clinical characteristics. The striking feature of the pre-operative history is its resemblance to the history of the ordinary fibroid case and the entire absence of any points in the history or physical examination which make it possible to suspect malignancy except in those cases in which the extension is so far advanced as to make operative cure or any other cure impossible.

In the first group, 2 patients only were living without recurrence, one 7, and one 4 months. One of these patients had the smallest tumor of the group, 2 centimeters in diameter; it was interstitial in location, without macroscopic evidence of infiltration, and it had the smallest number of mitotic figures of any of the tumors included in the group, 2,200 in the cubic millimeter. The other patient reported no signs of recurrence 4 months subsequent to operation. The tumor in this case was large, subserous, pedunculated, 17 centimeters in diameter, and adherent to the omentum. Not including the very small tumor just mentioned there were 2 cases in which the tumors were located interstitially; they showed no definite macroscopic evidence of infiltration beyond the uterine body. Both of these, however, recurred and were fatal.

As compared with other types of malignant tumors non-epithelial uterine tumors are said to be comparatively low grade in malignancy. This seems to be true in so far as metastasis in distant organs is concerned. In the present series definite indication of distant metastasis was not found in any case. But from the standpoint of rapid and extensive

infiltration these tumors must be classified as extremely malignant.

A comparison of the ages of the patients in the different groups of the series at the time of operation shows that as a group the patients with very malignant tumors averaged 50 years, while all the others averaged 40½ years. The patients included in Group 2, with the border-line tumors, averaged 41 years. A rapidly growing tumor at or after menopause is very suggestive of this type of malignancy.

Treatment. Five cases in this series were treated postoperatively with the X-ray and radium, but the after-histories indicate that no cures were accomplished in those cases in which the operative and microscopic examinations indicated extreme malignancy. One case (Case 18) is of special interest in this connection. The tumor microscopically belonged to the group of border-line or transition tumors having 800 mitotic figures to the cubic millimeter; at operation such extensive adhesions were found that the tumor could not be removed. The patient was given prolonged X-ray and radium treatment. After two and one-half years she is alive and apparently well with some regression of the tumor.

The present plan of surgically removing all uterine fibroids of appreciable size seems to be the procedure of choice. The low operative mortality figures which are shown by the work of skilled surgeons in the better hospitals are an argument in favor of the operative method.

In the present series of 72 cases there were no operative deaths. This remarkable absence of fatalities in a group of cases of so serious a nature must be looked on in a degree as accidental as is indicated by the usual operative mortality percentages in operations for fibromyomata.

Terminology. Several different names have been used to designate the malignant nonepithelial tumors of the uterus of the type discussed. Among these may be mentioned "sarcoma," "myosarcoma," "myoma sarcomatodes," "leiomyosarcoma," "myoma malignum," "malignant leiomyoblastoma," "malignant leiomyoma," and "malignant myoma." In the interest of a uniform terminology it seems well to use the term "malignant myoma."

SUMMARY

There is evident need of the establishment and recognition of histologic standards of malignancy in the classification of the nonepithelial uterine tumors.

In the present study the only single constant microscopic evidence of definite malignancy is the presence of large numbers of mitotic figures.

Many of these tumors have numerous large giant cells with multiple hyperchromatic nuclei which are often looked on as evidence of malignancy, but they do not contain mitotic figures. There is no evidence that such tumors are malignant.

In the less malignant tumors not containing numerous mitotic figures there are morphologic evidences of division of tumor cells by direct cell division.

Clinically the majority of patients with definitely malignant tumors present themselves for treatment about the climacteric or later. The tumors are difficult to distinguish in the earlier stages from ordinary fibromyomata; they are not cured by X-ray or radium, and the surgical removal of all fibroids of any appreciable size seems to be the best treatment.

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TREATMENT OF TUBERCULOUS OSTEO-ARTHRITIS BY BONE GRAFTS

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SURGEONS of a few years' experience have had occasion, as time has gone by, to review all the modifications, each one less mutilating, of the methods by which tuberculous osteo-arthritis has been treated. We have passed through the stages of amputation of the diseased limb above the lesion; total resection of the diseased joint—extirpated as one would extirpate a tumor—and atypical resection, the diseased portions being carefully sought out in the articulation and adjoining bones in order to extirpate them, interfering as little as possible with the cartilaginous line of growth and leaving the bony surfaces in shape thereby facilitating the formation of a new joint.

As regards the treatment in the majority of cases, we have today cast all these methods aside, but we should not overlook them when the patient has reached the fistulous stage with resulting destruction of bone and soft parts, when he is in a state of cachexia, and especially when he is an aboriginal in whom the lack of hereditary immunization does not assist in the localization of the tuberculous lesion, the basis of all conservative treatment. A study of the different operations which have been used has taught surgeons the pathology of the disease. It has demonstrated the various zones of the process and the marked venous congestion which exists in tuberculous epiphyses, lesions which are very clearly seen when presented in the pathological anatomy of the living subject.

Deep cauterization as an alterative for destroying the tuberculous foci, finally provoking cicatricial sclerosis of the lesion; the filling up of curetted cavities; and above all, the use of modifying subcutaneous injections which suffocate and silence the focus sometimes by surrounding it with connective tissue, at other times by surrounding it with fluid, thus making it more easy to draw off the products of the infection by repeated puncture—these are all processes which, we may say, introduce a new period in the history of the treatment of the disease.

The treatment with tuberculin—a biological process practically dominating all former methods—heliotherapy, and rest are today procedures which have proved very successful according to our statistics. These methods have placed an efficacious means of treatment in the hands of any who persistently follow a consistent program of application. The technique is quickly mastered even by

those who have had not much medical experience. All this is an advantage.

The present method of treatment is non-radical or non-surgical. However, since it requires 3 and 4 years to effect a cure, I have been interested in trying to discover some surgical means by which the time could be shortened.

To form a basis for judgment it has been necessary to study the exact causes of the local pathological processes and of the spontaneous cures produced by the more efficacious therapeutic procedures.

Let us first study the etiology and pathology of a diseased joint. The description I shall give is a little schematic, but it embraces the conception of the development of tuberculous osteo-arthritis as it is presented in the majority of cases of slow progress which are the more frequent.

I will touch briefly on the osseous formation of the epiphysis. The epiphysis is composed of spongy tissue, which is limited on one side by the cartilaginous line of growth and on the other by the articular surface. This surface is not entered by large vessels at any point but receives its blood vessels only from the periosteum covering the lateral and peripheral surfaces of the bone, which is reinforced by the capsule of the joint, ligaments, and tendinous insertions near which there are also blood vessels. Hence, when the capsule is overdistended by articular effusion, its vessels are also distended and thus because of their lessened lumen the blood supply to the epiphysis is diminished.

Throughout life the osseous system is constantly active in a struggle between the osteoblasts, which form bone, and the other elements peculiar to the marrow, the marrow cells and the myeloplaxes, the latter of which give way to the osteoclasts, destroyers of bone; and it is owing to the greater production of osteoblasts that the bone develops in the child. Later, in the adult, the osteoblasts and osteoclasts are produced in equal amounts. In old age, the bone becomes thinner, due to the excess production of the osteoclast. Success in the struggle sways from one side to the other in the different pathological states of the bone marrow.

The embolism which localizes in a bone and which generally comes from a primary focus, especially from a lymphatic gland, provokes a tuberculous marrow infection, that is, the bone marrow is sown with tubercular follicles which undergo mucous degeneration. Its elements become embryonic in type, and its peripheral capillaries are for the time being dilated while the central capillaries near the focus, in which abound tuberculous follicles undergoing caseous degeneration, are obliterated. These lesions produce a rarefying avascular osteitis in the trabeculæ of the bone, which is also toxic and partly due to the osteoclast.

The result in the competition of both systems—which, as I have said, are always active and in reciprocal action throughout normal and pathological life—is that the more rarefying the osteitis becomes the less is the intratrabecular marrow helped in its struggle to smother the invader; it is overcome and the tuberculous process gains ground, and therefore the alveoli, every day becoming larger, give better protection to the roots, as I may put it, of the granulations which, once the cartilage is destroyed or raised, penetrate into the synovial cavity sowing it with tubercles.

These predominant lesions of rarefying osteitis are accompanied by a very accentuated venous stasis and by a reduction of the blood supply through the epiphyseal arterioles, due to œdema of the periosteum, to a distention of the soft periosticular tissues and to lesions of mal-position following on reflex muscular contraction—causes which act more especially on the venous walls, which are more depressible than those of the arterioles, which are already contracted by the action of the tuberculous toxins.

To sum up, we have venous stasis, little flow of arterial blood, extravasated liquids, sluggishness in metabolism, lesions of malposition through reflex muscular contraction, and rarefying osteitis. All this assists in the formation of the granulations.

Let us see how the more efficacious therapeutic measures, rest and heliotherapy, act.

Rest, and here the best results are obtained from continued extension, acts as a depleting

Normal knee.

Tuberculous knee 10 days after operation.

agent by uniformly compressing, through the medium of the soft tissues, the articulation and the epiphyseal surfaces, facilitating the reabsorption of the extravasated liquids in the œdematous tissues which have undergone embryonic transformation. Therefore, it facilitates, by compression, the circulation of the blood, it lessens the venous stasis for this reason and because it eliminates the congestion that mal position has provoked through reflex muscular contraction. This contraction is overcome by continued extension.

Heliotherapy, apart from its action on the phagocytosis and its general tonic effect, acts as an alterative, since it is well known that the more the skin becomes pigmented the more efficacious is the treatment, and with this augmentation of pigmentation, it fills the part of the lampblack of the apparatus used in physical science, which allows luminous heat to enter and not to pass out when it loses the former characteristic becoming non-luminous. This alterative provokes dilation of the vessels, greater rapidity in the blood circulation and therefore reabsorption of exuded liquids which overwhelm the diseased tissues and which prevent an efficacious interchange of nutrition and the very effective phagocytosis in the struggle against the tuberculous process.

The cautery, by which Charcot has already cured several cases of spinal cord compression due to vertebral osteo arthritis, has proved a good method of replacing effective alterative processes. But this is not so far-reaching as heliotherapy and is more transitory and discontinuous; there is, moreover, always a danger of secondary infection.

Now, by therapeutic action we can facilitate an alterative procedure in the periphery of the diseased articulation, increase the arterial circulation and lessen the venous stasis, eliminate the malformations, increase the nutritive interchange and reabsorb the exuded liquids.

In this manner we help the normal tendencies of the organism to encyst, to sclerose Koerner's tuberculous follicles, the tuberculous unit, in whatever site or tissue it develops. That is to say, we are fighting against tuberculous myelitis, and if we find any means which fills this function and opposes a

rarefying osteitis, the pathogeny of which has been shown to be propitious in the progress of the disease, we shall have reached the point in our program which surgical intervention must complete in the spontaneous cure of the disease.

I have devoted much time in seeking some method of biological procedure to complete this program, and more than two years ago I decided on the use of the bone graft. I plan above everything to protect the tuberculous tissue, not to touch it, but to work at some distance from it, although I know that the graft will live even in caseous material and within the zone of tuberculous medulitis. Hugh McKenna, of Chicago, did not hesitate to curette the central spongy tissue of a spina ventosa, placing within the cavity an autogenous graft to fill it, and he obtained a successful result. In spite of this I have concluded that if I place the graft outside of the tuberculous tissue my results will be good, for the graft bed would be better here than if placed in the diseased tissue.

A bone graft is today the form of graft that gives the best results, for it is full of vitality, and is easily adapted to the form which we wish to give it, and we have complete control over it for several years.

In 1915, I was appointed official investigator on the theory of "Bone Grafts in Pott's Disease" to report my findings at the extraordinary session of the Society of Pediatry. Commenting upon 18 cases operated upon by me in my surgical ward, I gave the following conclusions as to the points in technique which my experience had taught me were essential to obtain successful results:

1. Autogenous grafts.
2. The most rigorous asepsis possible.
3. Careful preparation of the bed in vascularized tissues, obtaining a meticulous hæmostasis to avoid the formation of clots between the bed and the graft, for the clots would form a barrier preventing the capillaries from penetrating the graft.
4. Rapidity in transplantation, removing the portion to be grafted with a gouge only when the bed for receiving the graft has been prepared. A saw must not be used for removing the graft, since necrosis of the superficial layers of the graft would be caused by the heat produced by sawing and above all because of the production of bone saw dust which would obliterate the openings of Haver's canals, preventing the penetration of the graft by capillaries. The gouge must be well sharpened so as not to exceed the elastic play of the osseous trabeculæ when they are being compressed for resection.
5. No foreign body (suture thread, etc.) must touch the graft, which should rest to the greatest possible extent in the subcortical osseous tissue, that is to say not only below the periosteum but below the cortex of bone. However, it must never extend to the center of the marrow.

The subcortical bone fulfills these two last requirements: (1) that of vascularized spongy tissue and (2) that in process of forming new bone; whereas the cortical bone tissue is already more condensed. In the marrow there are two principal elements, one of vascularized and the other of fatty material, which later, on being traumatized during the severe operation of being compressed with the graft, give rise to large hæmatomata impeding or retarding the capillary penetration of the graft, producing necrosis of the graft, whereby it is reabsorbed.

6. Immobilization of the region operated upon. In the last bulletins of the society of surgery of Paris, for the sessions of 1918 and 1919, there appear the contributions from French and foreign surgeons giving the results of surgical experiences in the war, and I notice with satisfaction that they do not add a single detail to the conclusions formulated by me five years previously and which I have since confirmed not only in treating Pott's disease but in treating fractures, pseudoarthrosis or ununited fractures, etc.

Convinced of the effectiveness of the use of bone graft when properly applied, we will see how it can be utilized to accomplish our above mentioned purpose, taking into full consideration the new pathological conditions, such as are found in this disease.

1. Because of its trophic action, the graft has the virtue of producing in the surrounding area condensing osteitis, improving the power of latent ossification, which is especially

abundant in bones in which there is some inflammatory process such as tuberculous epiphysitis, which we know weakens when it is not assisted and ends in total rarefying osteitis. Hence it occurred to me to use a sheet of bony tissue placing it within the epiphysis. This would produce a condensing osteitis, and the lessening of the diameters of the osseous alveoli would, so to speak, suffocate the roots of the granulations which had previously taken ample hold but which under the new conditions would atrophy.

2. The graft, placed so as to extend from the diaphysis to the epiphysis without touching the articular cavity, would cross the cartilaginous line of growth and as Haver's canals of the graft are rapidly and abundantly penetrated by capillaries, they would serve to overcome the venous congestion and carry arterial blood to the epiphysis. We observe also that the perforation of the cartilaginous line of growth by the bone graft depletes the venous congestion. This, as I have said, is easily established for the epiphysis is practically isolated from the circulatory current, as its articular and diaphyseal faces, covered with cartilage, do not permit the passage of vessels and veins, which go out through the lateral parts of the epiphysis and then are compressed by the œdema of the periosteum. To produce and maintain an active, permanent, and aseptic deviation of the blood current—such as can be secured by means of heliotherapy and which is better than that produced by means of the cautery—and thus combat the epiphyseal venous congestion, I place two lateral grafts in the cellular subcutaneous tissue surrounding the articulation so that there are points of implantation in bony sites at both extremities of the grafts. These grafts are at once copiously penetrated by capillaries thus depleting the central congestion by the aspiration which this rapid peripheral circulation produces.

It is scarcely necessary to say that I do not contend that the grafts fill from the first day the varied rôle my conception assigns to them. Therefore, while they are growing, I continue for 3 months the application of extension, bandages, and weights, which have been put on 15 days before intervention so that it will not be necessary to perform the operation in the presence of an active tuberculous lesion. After three months the patient can get up, wearing light plaster bandages which keep the joint immobile.

During all this time, the lateral grafts have been growing and from the size of a toothpick they have become in 6 months of the breadth and thickness of a rib. I study the course of the disease by roentgenograms which show clearing up of the rarefying osteitis; and by palpation which shows that the granulations and the pain have also disappeared. At this stage I remove the subcutaneous lateral supports and within 10 or 12 days after this slight operation I turn the patient over to a masseur for treatment to bring back mobility to the joint. The joint is not very stiff as in the latter days play on the normal axes of the articulation has been possible, as muscular contraction no longer exists and the lateral grafts have molded their extremities into angular shape thus acting as a pivot.

In the first four cases of osteo arthritis I used the X-rays several times. I had to lessen the dosage after the first case, since the rays produced in it a radiodermatitis with ulceration of the skin that doubtless had an untoward effect on the bony and peri-articular tissues, as there was great limitation in the movements of the knee. However, the limb is in such a position that it makes a useful pylon.

I have given up the X-rays in my later cases and now am using moderate heliotherapy, more as a matter of hygiene and as a general tonic than for its local action. Although it is an aid, I do not consider it to be indispensable.

From the first days following intervention, an improvement can be noted, improvement in the general condition, increase in the color of the mucous membrane, disappearance of the yellowish tint in the skin, and a better appetite. These are all indications of the lesser quantity of tuberculous toxins produced and absorbed in the focus, and to which I called attention 5 years ago when treating upon Albee's graft in Pott's disease.

Since that time I have maintained that it is especially in cases of accentuated tuberculous

cachexia that operation is urgent, for there is produced at a distance from the tuberculous focus, as in breaking the spinous processes of the vertebræ, a traumatic irritation of the periosteum and the fractured body tissue, a process which is alterative, interfering with the venous congestion which exists in the tuberculous focus of the diseased vertebra, and brings about less production and less absorption of toxins.

PERTROCHANTERIC FRACTURE OF THE FEMUR

By ABRAHAM O. WILENSKY, M.D., New York

A SOLUTION of continuity of the femur, in which the line of fracture is situated at the angle of junction of the shaft and neck of the bone and passes through the great trochanter, is so uncommon that up to the present writing there have been very few authentic cases reported in the literature. A summary of the cases follows:

The first of these is described by Sir Astley Cooper in his book published in 1844. The patient was an old man and the fracture was sustained in falling. Union with good function resulted. Some time later, after discharge from the hospital, the man died of some intercurrent fever and the specimen of the fracture was obtained at the postmortem examination (Fig. 1).

The second case was described by R. W. Smith in 1854. The fracture followed a fall by a man of 70 years of age. An infectious process developed at the seat of fracture which resulted in death five days after the accident. The specimen of the fracture was obtained postmortem (Fig. 2).

Three cases are mentioned by Bennet in 1895. The clinical details are not well given; the illustrations are not very clear.

In the same year five cases are figured and described by Kocher. The name "pertrochanteric fracture" is employed for the first time. Previously the lesion had been called—and is still so called—by Stimson "fracture through the great trochanter and neck."

In the first of Kocher's cases the injury was sustained in falling backward and the line of fracture passed obliquely from the outer side and anteriorly, downward, posteriorly and internally. After healing, the neck of the bone protruded at a right angle from the shaft; except for this no other deformity can be distinguished in the illustrations. In the second case the injury followed a fall in which the man's body struck the ground on the side of the body opposite to the hip fractured. The line of fracture followed a very similar plane to that described in the first case, and the resultant deformity is almost identical.

Three other cases similar to one another are described. In each the mechanism is that of forcible extension in the dorsal direction in falling backward; and the line of fracture passes from above and in front downward, backward, and to the inner side. In healing, the lower ends of the upper fragments are all rotated forward on a transverse axis, so that a perceptible anteroposterior angulation is present at the point of union; a coxa vara is present owing to tilting up of the trochanters (Figs. 3 and 4).

A very excellent example of this fracture is present in the United States Army Medical Museum. I am indebted to the Surgeon General's Office for the following notes:

"While in a state of intoxication, was being assisted to his quarters by two comrades, pulled himself violently from them at the head of the stairs, and fell over the railing to the pavement below, a distance of 13 feet. He was taken to the hospital at once, when he was found to be suffering from concussion of the brain, in the stage of collapse. Reaction soon came on, when it was found that he had sustained an injury at the right hip-joint supposed, at first, to be a dislocation,

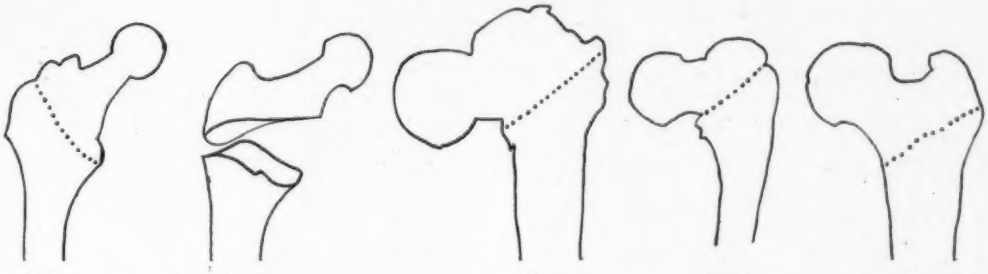

Fig. 1 Fig. 2. Fig. 3. Fig. 4. Fig. 5.

Fig. 1. Cooper's case.
Fig. 2. Smith's case.

Figs. 3, 4 and 5. Three of Kocher's cases. The X-ray photographs of Kocher's other cases as reproduced in his paper are not very clear.

but after manipulation under ether, was decided to be a fracture, the exact nature of which was not fully determined until after death, when it was found that a fracture extended through the trochanter-major and base of the neck of the femur, being partly extracapsular and partly intracapsular.

"..... It is believed that he struck the stone pavement on his right foot, producing both the fracture and concussion (Fig. 5)."

Stimson describes a personal case as being somewhat atypical in that the line of fracture "was very long and oblique, extending from the top of the trochanter downward and inward to a point ..... thought well below the lesser trochanter." The line of fracture was confirmed by exposure during operation. The patient, a man of fifty years, recovered with little, if any, shortening.

A case is mentioned by Gerster in his paper on the nail-extension treatment of fractures. The patient was 65 years old and healing of the fracture occurred in five months' time.

In a classification of 55 cases of fracture of the neck of the femur, McGlannan notes 3 as being pertrochanteric fractures. The patients were all laborers between 40 and 60 years of age and the fractures all healed. No other details are given and the X-ray photographs are not reproduced.

Below is the report of an additional case which, was recently admitted to my service at the Beth David Hospital.

The patient is a man of 65, who sustained the injury in falling on the street. Apparently he fell backward and landed on the side of his body and on the involved hip. There was immediate and total disability.

The first examination was made in the hospital one week after the injury. The limb was perceptibly shortened and markedly everted; the shortening amounted to between 3 and 4 centimeters. A large swelling was present over the outer side of the hip and, on palpation, the tumefaction seemed to consist of a much displaced trochanter. The most marked point of tenderness was at a point corresponding to the mid line of the thigh on a level with the fold of the groin. There was no ecchymosis of the skin. The tentative diagnosis was a fracture at the upper end of the shaft of the femur involving the trochanter major. The character of the visible deformity, the marked eversion, and the absolute loss of power indicated that the fracture was probably not impacted; the assumption was confirmed by the X-ray picture.

The roentgenogram (Fig. 6) showed the typical line of a pertrochanteric fracture. The fracture begins near the upper extremity of the trochanter major and passes downward, inward, and backward to terminate just at the lower margin of the trochanter minor; the line of fracture is parallel to the anterior intertrochanteric, or spiral, line. The fractured surface of the lower fragment looks upward, backward, and inward toward the median line; the corresponding surface of the upper fragment looks downward, outward, and somewhat backward owing to a tilt of the upper fragment. There is a sliding displacement of the shaft upward and outward so that the superior margin, or apex, of the trochanter major has moved obliquely upward and outward for more than an inch from its relative normal position while the fractured surfaces still remain in contact for their greater parts. There seems to be no subsidiary lines of fracture. The texture of the bone does not indicate any excessive amount of rarefaction, perhaps not even as much as one would ordinarily expect to find present in a man of the patient's age. There are no other lesions present in the immediate neighborhood demonstrable in the roentgenogram.

At my request the fracture was reduced and immobilized under traction by my associate Doctor Brody. Immobilization was effected by a plaster hip spica including the leg and foot. This method was preferred to an extension apparatus because it was feared that the latter would not work efficiently with this particular patient. After two months the cast was removed and an X-ray examination demonstrated that the fracture had united firmly and with a large amount of callus. Within a comparatively few days the man was able to elevate the leg actively while lying in bed and, thereafter he was permitted to walk with the aid of crutches. These, also, were quickly discarded and at the end of three months the man walked very well but with a slight limp. The residual shortening amounts to between 2 and 3 centimeters.

The anatomical end-result is shown in the accompanying roentgenogram (Fig. 7). The shaft of the femur rides higher than usual, but owing to the fact that the fractured surfaces are in contact, and that during the process of healing the latter remained parallel to one another there is no other anatomical abnormality. The angle between the neck and the shaft of the bone has, for the latter reason, not been disturbed and no true coxa vara deformity can be distinguished in the X-ray photograph; it is likely that this normal relationship will continue permanently because of the abundant callus not permitting any bending of the neck and any resulting lessening of the angle between the latter and the shaft. The broadening of the total transverse diameter of the hip is incidental and abduction is limited thereby to an inconsequential degree because of the outward flare of the ilium to which the tip of the trochanter is opposed, and by which motion in abduction is limited; the extent of the limitation is proportional and equal to the diminution of the arcs of circles as one progresses from the lower border of the flare of the ilium upward and outward to its crest.

We were all much impressed with the rapidity with which the fracture united and with the relatively large amount of callus which formed. If nothing else this would seem to distinguish this variety of fracture from those of the neck of the femur. In the latter, when the fracture is not impacted, healing, when it does take place, is tedious and prolonged; nor does one see such a relatively large amount of callus. Fractures near the base of the neck and in the trochanters may be entirely extracapsular while those in the neck proper usually open the joint capsule for a variable distance or are intra-articular. The significance of the latter complication on the want of, or on the character, the extent, and the rapidity of cicatrization and ossification seems settled and the majority opinion leans toward the assumption that the open communication of the fracture with the interior of the joint has no material effect on the progress, or lack of progress, of union of the fractured surfaces. With the neck fractures it seems that the difficulty lies in an adequate and efficient reduction and fixation of the opposing fragments in an anatomical position favorable for healing; in the pertrochanteric fractures the possibilities of attaining this purpose are much more easily accomplished. A most important reason for the frequent non-healing of unimpacted fractures of the neck of the femur is to be found in a relative minimum blood supply in the head and neck of the bone, as compared to a relative abundant blood supply in the trochanters and in the upper part of the shaft.

In all of the reported cases of pertrochanteric fracture the injury followed a fall. In all, except one case, the fall was directed backward; in attempting to recover the normal balance from the relative hyperextension of the lower limb at the hip-joint, an extraordinarily powerful pull is directed at the upper end of the bone in which all the lines of force converge and are concentrated in the plane corresponding to the lowermost limit of attachment of the Y ligament of Bigelow; the fracture necessarily results at this level. In the exception—one of Kocher's cases—the fall was directed toward the side of the body away from the implicated hip. The mechanism must necessarily include a powerful leverage by adduction of the entire lower extremity, a fulcrum and point of resistance having first been established by fixation of the upper end of the bone by the Y ligament; again the fracture must occur in the same plane. In our case the man fell backward but, in falling, the body turned so that he landed on his side and on the hip. In addition to the hyperextension the mechanism probably included a twisting force which found its minimum resistance in the line of fracture. I am rather inclined to believe that the direct impact of the hip against the ground had little to do with the fracture; I am led to this opinion from a consideration of the mechanisms in the other cases.

Fig. 6. Taken before reduction and immobilization.

Fig. 7. Two months after immobilization.

The Y ligament of Bigelow seems to be one of the most important factors in the production of a pertrochanteric fracture. Of all the structures in the immediate neighborhood of the fracture, this ligament is the strongest and most unyielding; in all the varieties of fracture and dislocation—and the latter injuries are most severe and are caused by extreme degrees of violence—it is found intact. So dependable is the ligament that all the methods of reduction of dislocations of the hip are built upon the theorem that the structure always remains intact and can be used as a fulcrum by, and upon which, the end of the bone can be levered into place. In producing the fracture under discussion it seems most probable, as indicated previously, that the injury results from a play of force in which an extraordinary overextension of the trunk at the hip-joint occurs in the dorsal direction with, and upon, the lower limb as a relatively fixed pivot; an enormous strain is transmitted through the unyielding Y ligament which, while incidentally aiding the muscles very powerfully in fixing the upper end of the bone, at the same time determines a line of greatest weakness and least resistance in the bone. In attempting to recover the balance the long flexor muscles of the thigh are very strongly contracted and a powerful stress is exerted across the length of the femur which has the tendency to bow the femur in a forward direction. A sufficient continuation of the indicated stress and strain results in a solution of continuity, and the latter must necessarily take place at the line of least resistance, determined by the Y ligament, which corresponds with the lowermost line of attachment of its fibers a little below the junction of the neck and shaft of the bone at approximately the linea aspera. That is the typical line of a pertrochanteric fracture.

Mathematically considered the plane of greatest weakness in the femur corresponds accurately with the plane of atypical pertrochanteric fracture. Mechanically, the anatomical structure in the neighborhood of the hip, and including the thigh and trunk, is essentially that of a cantilever beam : a relatively rigid upright, *AB* Figure 8—the shaft of the femur—supports at its upper end one extremity, *A*, of a beam, *AC*, fixed at an angle, *BAC*—the neck and head of the femur—and at the opposite end, *C*, of the latter a weight—the trunk—is carried. The force constantly exerted at any point of the beam—technically known as the "bending moment" and which in our discussion would correspond to the force necessary to produce a fracture at any given point of the beam—is equal to one-half of the weight carried, multiplied by the perpendicular distance between the point of application and the point of fracture of the beam. In the given instance the weight is carried at the extremity of the

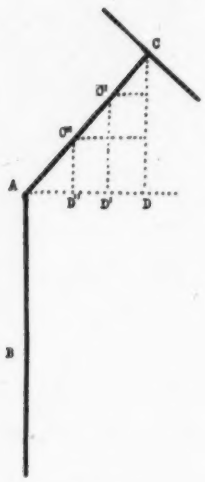

Fig. 8.

beam, C, and the perpendicular distance (l) between the point of application and point of support corresponds to the length AD. Algebraically it is indicated as follows:

$$M = \tfrac{1}{2} W.\, l \qquad (1)$$

in which $l = AD \qquad (2)$

$$\therefore M = \tfrac{1}{2} W.\, AD \qquad (3)$$

At any given two points on the beam C' and C'', in which C'' is nearer to the point of support of the beam, the bending moment exerted from the point of application of the force at C grows progressively greater the nearer the point of fracture, C' or C'', approaches the point A which is the point of support of the beam. This is so because

For C' $\quad l' = DD' \qquad (4)$

and for C'' $\quad l'' = DD'' \qquad (5)$

$$DD' < DD'' \qquad (6)$$

and if the values are substituted in equation (1) given above we obtain

for C' $\quad M' = \tfrac{1}{2} W.\, DD' \qquad (7)$

and for C'' $\quad M'' = \tfrac{1}{2} W.\, DD'' \qquad (8)$

$$DD' < DD'' \qquad (9)$$

$$\therefore \tfrac{1}{2} W.DD' < \tfrac{1}{2} W.\, DD'' \qquad (10)$$

or $\quad M' < M'' \qquad (11)$

Progressing further it follows that the point A must necessarily be the point where the "bending moment" exerted from the point C is at its maximum—in other words where the structure is at its weakest. This is so because

for C' $\quad M' = \tfrac{1}{2} W.\, DD' \qquad (12)$

for C'' $\quad M'' = \tfrac{1}{2} W.\, DD'' \qquad (13)$

and for A $\quad M''' = \tfrac{1}{2} W.\, AD \qquad (14)$

$$AD > DD'' > DD' \qquad (15)$$

$$\therefore \tfrac{1}{2} W.AD > \tfrac{1}{2} W.DD'' > \tfrac{1}{2} W.DD' \qquad (16)$$

or $\quad M''' > M'' > M' \qquad (17)$

Dispensing with the technical terms and applying the result to the anatomical problem the calculation indicates that the weakest part of the support furnished by the thigh lies at the point of junction of the neck and shaft of the femur.

As regards the position of pertrochanteric fractures in the general classification of fractures of the femur, it seems to me that Cotton takes the best view. All fractures in the general neighborhood of the trochanters and the base of the neck are made to conform to one of three types. Types A and B are complicated forms of intertrochanteric fractures with subsidiary lines of fracture running in various directions. Type C corresponds to what is described in this communication as a pertrochanteric fracture. All of these types have the valuable characteristic of healing promptly and efficiently.

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THE ORIGIN OF TUMORS OF THE OVARY

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THIS article is but a résumé of a larger work which will appear shortly in monograph form. A review of the literature involves a colossal work and can be referred to only in a very cursory manner in a contribution of this nature. Suffice it to state that the more one delves into the subject the more is one confused with the multiplicity of views and the number of structures from which tumors within the ovary may arise. Some authors have dealt with embryological studies of the ovary, and others have given their attention to determining the origin of tumors, but seldom has any one author taken up the second portion after having acquired a good knowledge of the first by his own research. It is quite impossible to determine the origin of epithelial tumors, unless one knows the structures from which these tumors may arise.

It is my purpose first to describe the epithelial structures found in the normal ovary, and having determined this origin and development then to pass on to consider the origin of tumors.

The work then divides itself into two headings:

1. The origin of epithelial structures in the ovary.
2. The origin of epithelial tumors of the ovary.

I. THE ORIGIN OF EPITHELIAL STRUCTURES OF THE OVARY

It is impossible to understand the reason of many epithelial structures found in the adult human ovary except by a careful and painstaking study of the human embryo at various stages of its development. Human embryos, well preserved and fit for serial sections, are difficult to obtain, and after most careful study of those at my disposal I had to have recourse to comparative embryology to find the origin and nature of many epithelial structures. Comparative embryology has not only the advantage of material easily obtained in a fresh state, but also the advantage of presenting the phenomena of ovarian development in a much clearer sequence of changes. That these phenomena are the same in the higher vertebrates and in the human species cannot be doubted.

This work is based upon sections both serial and non-serial from ovaries of 127 individuals of all ages from a few hours to 80 years, as well as upon serial sections of five human embryos. The comparative study covers roughly 15,000 sections, mostly serial, from the cow, pig, sheep, cat, mouse, rabbit, guinea-pig and the dog and from embryos of the cow, dog, pig, and cat.

It would be impossible to overemphasize the importance of serial sections. In many instances my most obvious conclusions, drawn from the study of a single slide, suffered a complete contradiction by a mere glance at the neighboring slides in the series, and it is possible readily to realize that the conclusions of many authors would never have been put on paper, had serial sections, and not single slides, been at their disposal.

For purposes of comparative study leading to a better knowledge of human histology and embryology, there are no two other domestic animals so useful as the cow and the dog and their embryos, and since it was the generative organs of these two which led to this research it will best serve my purpose to confine descriptions to these species, describing others only when corroboration or elucidation are necessary.

A careful study of the mammalian ovary leads at once to the important fact that from a developmental point of view the resemblance between the ovary and the testis is a very striking one. There is nothing in the testicle that has not had at some time in embryonic life its homologue in the ovary. It therefore follows that of the structures that

are permanent in the testicle, some are transitory in the ovary and disappear completely, others degenerate but do not disappear, still others, remain but are modified to suit a modified function. The female embryo having developed more or less perfect testicles, progresses still further by destruction, modification, and addition. It is the development of all the testicular elements and their subsequent incomplete atrophy which has made of the ovary such a fertile field for the development of epithelial tumors of the most varied types.

What makes the ovary of one mammal differ most from the same organ of another mammalian lies in the amount of atrophy which ensues after the ovary has reached its full structural development. Of all the species examined, the cow, the bitch, and the cat suffer the least amount of atrophy. Hence, for this very reason they are exceptionally convenient for developmental study.

Until recent years it was almost unanimously thought, chiefly due to the teaching of Waldeyer, that the germinal epithelium of the ovary never penetrated farther than the outer cortical third of the organ. Whatever glandular structures were found within the hilus or medulla of the ovary were thought to be due to the invasion of the ovarian stroma by the tubules of the wolffian body. The wolffian body or mesonephros was consequently divided into two portions: that which invaded the ovary was called the genital portion, whereas that outside the boundaries of the ovary was called the urinary portion of the wolffian body.

Comparative study of adult ovary. If we examine almost any section from the ovary of the bitch, we find an unexpected wealth of epithelial structures. These, at first, seem to be without order and of several types. In the heart of the ovary, frequently in the midst of the great blood channels, we find the rete ovarii.

The rete ovarii. This structure corresponds to the rete of the testicle. It is an irregular tortuous gland-like structure, with numerous folds of its mucous membrane, which is lined for the most part by a columnar epithelium. The structure in some animals suggests a low grade of malignant carcinoma. The cavity of the rete varies much in size in different species. In my series the sheep present a very small rete ovarii, ofttimes difficult to find in the center of the ovary. In the dog and cow it is a large structure, often extending almost from pole to pole of the ovary as it passes on its tortuous course between the main blood vessels.

In the human the rete ovarii varies considerably in size in different individuals and even in the two ovaries of the same individual. In one of my cases one ovary is a mass of fœtal remnants without ova, whereas the other contains thousands of ova without any trace of fœtal remnants except the diminutive rete. We must look upon the rete as a fœtal remnant, and its atrophy and complete disappearance is quite the exception. In my numerous specimens a careful search has always resulted in finding a rete though at times it is quite diminutive and might readily be overlooked. But in several of the ovaries from the sheep I have not been able to find a trace of it.

The medullary cords or rays. In the ovary of the bitch there are numerous solid cords or tubules which course in a very irregular manner from the cortex to the center. Their arrangement generally speaking is radial though very irregular and convoluted. In the multiplicity of cells which line them or compose them, one may distinguish three dominant types.

The lining which conforms with the first type, consists of tall shaggy cells with a wide base and shrunken body prolonged into a thin protoplasmic filament which not infrequently passes across the lumen to join with a similar process from a cell of the opposite side. The nucleus is near the base. This type of lining is found chiefly in the tubular rays. At times a fold starting from the periphery of the tube, causes on transverse section an appearance suggestive of a polypus projecting into the lumen. This is also covered with a similar type of cell and the fibrous core, usually hyaline and without nucleus is joined by a thin velamentous fibrous tissue with the connective tissue at the periphery of the ray.

The second type is found in the solid cords. The dominant cell is syncitioid in type and in its functionless state resembles very closely the syncitioid cells of a young placenta that has been retained *in utero* for a short period after loss of function. The third type is less commonly found and resembles a true glandular duct, lined by cubical epithelium. These three types when traced through serial sections pass at times almost imperceptibly the one into the other. At other times the change is abrupt especially after an acute bend or tortuosity of the tube.

In serial section many of the rays are found to communicate at one end with the rete ovarii. The distal end of the ray is lost in the periphery of the ovary.

Development of the epithelial tissue of the ovary. I will not go into details of length and age (probable) of the various fœtus. These matters have little or no bearing upon the subject. The first sign of the development of the genital organs is found in a heaping up of the epithelium at a spot on the genito-urinary ridge. Immediately under this, at this early stage, lies the wolffian body with its ducts and glomeruli. This heaping of epithelium constitutes the germinal layer or the germinal ridge. Its origin, so far as is known, is from the peritoneal or mesothelial surface, and it lies to the inner side of the mesonephros. (A second genital ridge appears slightly later to the outerside of the wolffian body. This eventually gives rise to Mueller's duct. Slight confusion arises here owing to the varied nomenclature used. Some authors apply the term germinal epithelium to the whole covering of the wolffian body, while others restrict it to that portion which gives rise to the ovary. I shall use it in this restricted sense.) This is the beginning of the genital gland—the testes or ovary. Soon this epithelium begins to burrow and to arrange itself in columns coursing more or less at right angles to the surface, and running toward a narrow hilus. During this development the ovary has gradually become pedunculated.

If we now turn our attention to the embryos of the cow at various stages of their development, we find that at one time the ovary and testicle form a pedunculated structure which occupies the inner side of the wolffian body. To the outer side lies the differentiated tissue which is destined to form Mueller's duct, the forerunner of the fallopian tube. Between these two structures lies the large wolffian body with a wide base of attachment posteriorly. Its main excretory duct, the wolffian duct, lies slightly to the inner side of Mueller's duct. From the inner surface of this excretory duct many tubules take origin, then run along the outer surface of the wolffian eminence, then they bend suddenly upon themselves, their lining epithelium begins to stain more readily with eosin, the cells become much larger and the tubules themselves become very much twisted and finally end in a glomerulus, near the inner surface of the wolffian body. A small artery issuing from the main arterial trunk in front of the vertebra passes into the wolffian body at its attached border, courses along its inner free margin to end in the vascular tuft of the glomerulus. Separated by only a very short distance from the uppermost glomeruli, the fibrous tissue of the ovary takes its origin from and becomes continuous with the fibrous tissue of the wolffian body. The glomeruli are more or less arranged in a single row and therefore, their convex surface, where it is pierced by the glomerular artery, lies in toward the ovary. Except at both poles of the ovary, the space between Mueller's duct and the hilus of the ovary is quite wide and rounded, filled in by the enormously developed tubules of the wolffian body. So far no differentiation of ovules is visible. The cells of the whole ovarian surface seem to be uniform. They consist of deeply staining cells which resemble more or less ordinary lymphocytes. In an embryo of slightly more advanced age, we find remarkable changes have taken place. The growth of cells from the germinal layer of the ovary is no longer restricted to the immediate surface of the ovary, but has penetrated into the medulla and fills the hilus, and, separating the fibrous tissue which seems to form a capsule for them, they pass as deeply staining branching cylinders of tissue out of the hilus and along the wolffian body between its

free border and the glomeruli. These cylinders gradually grow smaller in extent as they travel along the wolffian body to end at a point near the free border of the mesosalpinx between the outer pole of the ovary and the fimbriated end of the tube. In their passage along the wolffian body they enter into intimate contact and become continuous with the cavities of at least two and may be as many as six of the glomeruli of the wolffian body. They are separate and distinct from the wolffian duct throughout their whole course, and enter into contact with it only through the interposition of the tubules and glomeruli of the wolffian body. There is, therefore, direct continuity between the surface epithelium and the wolffian duct through the medium of the medullary cords or tubules, the rete ovarii, the wolffian glomeruli tubules and duct.

So far as my experience goes, in no other animal can this be studied to such advantage as in the embryos of the cow. At this stage there is no differentiation of the ovarian epithelium tissue into tubules or ducts. It presents a deeply staining cellular structure, the cells of which possess a minimum of cell protoplasm with a deeply staining nucleus like that of a lymphocyte. Nothing could be more striking than the contrast between this darkly stained band of germinal epithelium and the adjoining tissues of the wolffian body. The latter is made up of large cells with a large amount of protoplasm which stains very deeply with eosin. At this stage of development, primordial ova are distinguished within Pflueger's tubules.

Slightly deeper toward the center of the ovary, in what I may designate as the second zone, or zone of medullary rays, solid and hollow strands, continuous with Pflueger's tubules, present themselves with primordial ova in their lumina. These are the forerunners of the medullary cords. Still farther toward the center these tubules in the medullary cords pass insensibly into a richly tubular structure rete ovarii which passes out of the ovary to pursue its course, which I outlined above, and to enter into contact with the glomeruli and glomerular ends of the wolffian ducts. In short, what was formerly a mass of darkly staining cells slowly and insensibly differentiates itself into tubules, by that same process by which Pflueger's cords differentiate themselves into Pflueger's tubules and the medullary cords into medullary tubules. In the ovary, as in the testicle, the continuity, therefore, is complete from the surface epithelium of the ovary to the wolffian duct. The parts nearest the germinal epithelium have to do with the generation of the reproductive elements, the remoter portions are the conduits along which the ova would travel if they pursued the same course as spermatozoa in the male. But in the female, ova are cast off into the peritoneal cavity and the efferent tubules become useless and atrophy completely or in part. Are all these parts maintained in their integrity through fœtal into postnatal and into adult life? The answer is decidedly in the negative, and it is the atrophy which ensues in the ovary owing to the ova pursuing a different route of migration which has led to so much confusion. A study of the adult organ cannot give any connection whatsoever between these various parts of the one and the same system owing to the complete atrophy of intervening links.

It must not be assumed that the extra-ovarian portion of the canals reach as complete a development in all other species as it does in the embryo of the cow. For example, in the human the extra-ovarian portion of the efferent ducts is relatively small and atrophies easily, so that only that portion remains (for some time) which is necessary to effect a junction with the wolffian ducts. Such were the conclusions to which my work led me, namely, that there are no parts in the testicle which have not their homologous parts at some time in embryonic life in the ovary.

These homologous parts may be clearly represented thus:

TESTICLE	
1. Seminiferous tubules	Of germinal epithelial origin
2. Vasa recta	
3. Rete testis	
4. Efferent canals	Of wolffian body
5. Vas deferens	Of wolffian duct

OVARY

1.	Pflueger's tubules	Of germinal epithelial origin
2.	Vasa recta or medullary rays	
3.	Rete ovarii	
4.	Kobelt's tubules of epoophoron	Of wolffian body
5.	Gaertner's duct	Of wolffian duct

If we trace the course of the germinal epithelium from its surface to the wolffian body, it could be represented as in diagram.

The first portion of the downgrowth represented by *a*, *b*, and *c* constitutes the medullary ray. The distal or cortical portion, *a*, atrophies easily and disappears completely. The second portion, *b*, becomes the normal graafian follicle. The third portion, *c*, atrophies more or less and may or may not be absorbed. From the lower portion of the rete several tubules finally merge into one which passes out of the ovary to unite with the glomeruli of the wolffian body.

To recapitulate: it has been shown through embryological studies that the ovary at some time in its development possesses a system of tubules, which have their exact homologues in the seminiferous tubules and excretory ducts of the testicle. Embryological studies have also shown that all the structures within the ovary, namely, Pflueger's tubules or cords, the medullary cords and the rete ovarii, to well outside the boundary line of the normal ovary, are of germinal epithelium origin; and it has been demonstrated that the tubules of the epoophoron, or of the wolffian body, do not penetrate into the ovary, but are met outside the ovary by the rete ovarii as it passes out of the hilus.

I now wish to take up other facts which have heretofore not been recognized and which offer incontrovertible arguments for the substantiation of the conclusions stated above.

In the first place, I have found so frequently immature ova within the whole length of the medullary cords, that there can be no doubt that they arose *in situ*. At a certain stage in the development of the ovary, it is not the exception to find this condition of affairs. Their presence is transitory and their life history is a short one. As we will see later on, this is the fate of thousands of these ova. They do not all perish.

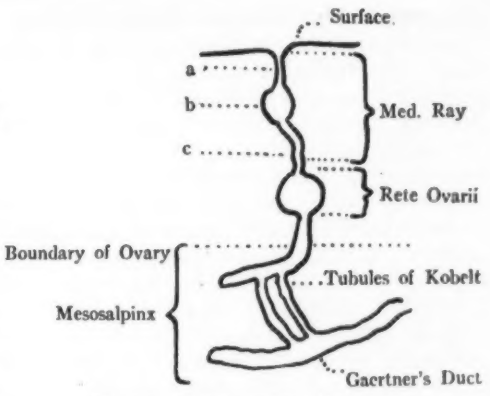

Diagram of development of the ovary.

Occasionally an ovum seems to escape. But it is a fact that where one ovum comes to maturity, a thousand are lost in the process. The same statements apply to the rete ovarii, though in a much more restricted sense. In all my specimens of embryos I have found about ten or twelve ova right in the center of the rete. Inasmuch as these very soon succumb to the general destruction, and as ova have no motive power of their own, and as the character of the tubules would preclude any possible motion being imparted to the ovum, we are forced to the conclusion that this again is incontrovertible evidence of development of ova from the lining both of the medullary cords and of the rete ovarii. Is it not also incontrovertible evidence that these structures are derived from the germinal epithelium, and have retained some of their powers which were impressed upon them in their primitive state? To the arguments of those who claim that both the medullary cords and the rete ovarii are derived from the epoophoron, I can only add further the statement that it seems quite inadmissible that nature would allow so important a function as oogenesis to be shared by two structures of totally different origin.

Have these fœtal rests any functions? Without the slightest hesitation one can say that in the human ovary they have no normal function. There cannot be the slightest doubt but that the only normal function they could perform would be the function of

oogenesis. But we know equally well that the formation of ova is a function of division and differentiation which in the human species takes place exclusively during fœtal life and the first two years of postnatal life. Scientific opinion upon this point is quite unanimous. But there is not the same unanimity when it comes to the question of the other mammals. In the lowest members of this order, I think it is generally admitted, oogenesis goes on, not only in fœtal and early postnatal years, but throughout the whole of the sexual life. I, myself, am convinced that ova are developed in the bitch throughout her whole life, and in the case of the cow at particular periods. If such is the case, from what do these ova originate? From a careful study of the bitch's ovary, the different types of tissue which I have described above as being found in the medullary tubes are but different stages in the develop ment of ova. In the second type, where the tissue presents much the character of syncytial cells with vacuoles, it is but the beginning of that development which eventuates in the formation of ova. I am convinced that this goes on throughout at least the sexual life of the bitch, and that ovulation by its stimulating influence, or by the consequent mere increase of vascularity awakens these oogenitic factors temporarily into activity. I have found ova in all stages of formation. In the vast majority of these developments the ova are quite imperfect and are doomed to a short life. Hundreds of them in one section may be seen as the mere phantoms of ova. If the ovule has developed but slightly (it will have developed a vitelline membrane early in its career) and then succumbs, it will be found with its vitelline membrane somewhat distorted and surrounded by a mass of follicular cells of deeply staining syncytioid characters. If development has gone on further before retrogressive changes set in, the syncytioid cells lose that character and take on the character of the first type as described above, that is, the ovum, composed now usually only of a vitelline membrane filled with degenerative droplets, is surrounded by a layer of tall, delicate, slender, filamentous cells which stain poorly. This is the prototype of the zona radiata of the discus proligerus.

These facts taken singly or together cannot leave any doubt as to the germinal origin of all the tubular and solid epithelial structures found in the subcortical and central zones and hilus of the human ovary. For if such is the case with the higher vertebrates, we cannot but believe that the process is the same in the human species. It seems quite impossible that two tissues so widely different as germinal epithelium on the one hand, and wolffian ducts on the other, should possess such high potentialities and specialization as are required to develop ova and their necessary adjuncts. True, if we go back far enough, both germinal epithelium and wolffian ducts were derived from the same cells of the cœlomic cavity. But with that type of argument we need go back but a little further to realize that all tissues of the body have common parents in an ovum and a spermatozoon.

The atrophy of structures so far described refers only to those superfluous tissues of fœtal origin, but in the dog and in several other animals there takes place a second downgrowth of germinal epithelium into the cortex of the ovary. This usually takes place in adult life. The invasions are quite glandular in appearance, never contain ova and are fruitful sources of new-growth with lower animals. Whether such a second invasion takes place in the human has never been determined.

In reference to this second invasion of the cortical zone by the germinal epithelium in postnatal or adult life in some animals Winiwater states: "An important fact remains, we never find in this glandular invasion nuclear elements so characteristically found in germinal cells on their way to become ova. So far as my experience goes this late invasion does not produce ova."

I am convinced that these late characteristic invaginations of the germinal epithelium, at an age when the animal has reached, or almost reached, full development, offer a ready explanation for that strong tendency in the human ovary for the germinal epithelium when invaginated by any of the

causes which will be mentioned later, to burrow and invade deeply and give rise to a typical growth. Waldeyer vaguely gave expression to the same opinion when he stated that this new formation of tubules from the germinal layer in some of the lower animals was but an expression of an inherited tendency in these cells.

THE INTERSTITIAL CELLS OF THE OVARY

In order to avoid confusion, it may be well to state that according to recently accepted views there are two distinct types of cells in the stroma of the ovary. It will not do to speak of them in common as interstitial cells, or as stroma cells, or as connective-tissue cells, because they have distinct separate origins which bind the one on the one hand to the epithelial group, and the other to the connective or fibrous tissue group. The former is of mesothelial, the latter of mesoblastic origin. However, *faute de mieux*, the term interstitial cells has passed into common usage, and as a means of distinction the mesoblastic tissue is known as the connective tissue or supporting tissue, or stroma.

Since Linion's and V. Winiwater's articles, an almost infinite number of works have appeared, culminating in Miss Lane Clappon's and Miss McIlroy's epoch-making article. With a lucidity that deserves the highest praise they have shown that there are two distinct cells in the ovary, the interstitial cell allied to and originating from the germinal epithelium and the connective-tissue cell proper derived from the connective tissue of the wolffian body. The former, therefore, possesses much of the potentiality of the oogenic tissue, the latter is but a supporting structure. My work upon the human ovary tends in every respect to confirm their work upon the mammalian organ. My specimens fortunately contain three of exceptional importance in this connection. Two of them are from the human ovary, one of a fœtus of about 7½ or 8 months, the other a child which died suddenly immediately after birth owing to a diaphragmatic hernia. In each of these specimens, whether due to some peculiarity in the stain, or whether due to the phase of development of these structures, each interstitial cell is so clearly defined and so beautifully stained that it offers the greatest contrast to the slender fibrillar intervening connective tissue. Their origin from the germinal epithelium stands out most clearly, and their differentiation from the follicular cells is as yet not complete. The limit of invasion of these cells in the ovarian tissue is clean-cut, and here again offers the most marked contrast to the faintly-pink-stained connective tissue.

The third specimen is from a rabbit, which had its back broken twenty-four hours before parturition by being clumsily lifted by the neck by one of the orderlies. Immediately paralysis of the hind legs and rear portion of the body set in. Parturition set in next morning, and the rabbit died in the second stage of labor without being able to expel the fœtus. The ovaries in this case were cut. They are from pole to pole just one mass of lutein cells. The outline of the true corpora lutea is well marked by a delicate band of fibrous tissue. Between them and all about them right down to the depths of the hilus, similar cells fill the whole field. Every interstitial cell in the ovary seems to have been converted into a large oval or polygonal cell resembling in every particular but one, the true lutein cells of the corpora lutea. That one and only distinction consists in a slightly smaller size of the extracorpus cells. Most minute examination with the highest powers of magnification reveals no other differences. The fine strands of fibrous tissue that interlace with these cells are readily seen. With Sudan III the whole ovary seems a mass of fat.

There can be no doubt that the ovary contains two types of cells, the interstitial and the connective tissue; the former, derivatives of the germinal epithelium and capable of transformation of form and endowed with secretory function; the latter merely supporting structure in the strictest sense of the term.

II. THE ORIGIN OF EPITHELIAL NEW-GROWTHS OF THE OVARY

The work in the preceding pages has led to a generalization the bearing of which is far-reaching, namely that all epithelial struc-

tures within the ovary are derived from a common source—the germinal epithelium.

Division and differentiation has led to wide differences in form and function. But we have seen that the characters—the primitive characters—of this epithelium may make themselves manifest when the proper stimulus comes, and we have seen how imperfectly that assumed function may be performed. This assumption of power is capable only by virtue of the inherited susceptibilities of these cells which are among the oldest in the animal kingdom. With this knowledge afforded by comparative and human embryology, the subsequent discussions will be greatly facilitated.

It would seem at first sight as if we stood today just where we were twelve years ago, as regards the histogenesis of simple and malignant tumors of the ovary. The various classifications which have been given have not as a rule been histogenetic, but rather, topical, clinical or histological.

Under such systems, the varieties become as a rule (except perhaps the first) so numerous that the subdivision becomes cumbersome. Clinical divisions and pathological subdivisions of any general system have become almost a necessity, but the wholesale multiplication of varieties has much in it that deserves condemnation.

I have tried to give a histogenetic subdivision which sacrifices nothing to brevity.

A. Tumors arising from the germinal epithelium { Ovulary. / Non-ovulary.
B. Tumors arising from the stroma.
C. Tumors due to cell inclusions.

"It would be very fine," writes Pfannenstiel, "if when studying each tumor separately, we could place it in its proper histogenetic category." I quite agree with Stratz, that a subdivision of tumors into cystic and solid, is not only unscientific but also impracticable, for neither clinically nor anatomically do tumors allow themselves to be placed in either one of these classes.

What are the structures in the ovary from which epithelial tumors may arise? They are germinal epithelium, follicular epithelium, ova, medullary cords, rete ovarii, corpora lutea, corpora atretica and the interstitial cells. If we include also inclusion tumors, we have covered the whole of the tissues from which they can take origin. But if we ask, can tumors arise from each of these? then the matter becomes one in which you form your own conclusions, either from mere preponderance of opinion or from your own observations. However, it seems advisable to deal with the subject from the view-point of these fixed and known tissues, rather than to take up each class of tumor and try to work back to its origin.

TUMORS ARISING FROM THE GRAAFIAN FOLLICLES

Wendeler very aptly remarks that it was natural, owing to the cystic formation of most tumors, to suppose that they were derived from follicles.

Can the epithelial lining of the graafian follicle give rise to tumors? In the first place, a distinction must be drawn between the membrana granulosa of a developing graafian follicle and the single layer of partially developed epithelium which surrounds a primordial ovum in the quiescent state.

If we exclude the condition which is commonly known as "hydrops folliculi," I can say with every emphasis, that I do not believe that tumors can arise from the graafian follicle in process of development or degeneration. For years the pathological material from several hospitals, both abroad and at home, has been examined with this end in view, and the result of my observations cannot be expressed in other terms. The varieties of degeneration of the membrana granulosa before or after the death of the ovum are few. The process is singularly simple, and it has never fallen to my lot to see these cells take on characters such as would justify one in assuming that they form the lining of a true cyst.

Are we to classify as tumors the condition known as "hydrops folliculi"? It seems to me that it is a misapplication of a term. Is it even a pathological state? It has happened so often that the small cystic ovary, or fibro-

cystic ovary so called, has yielded under the microscope a prolificity of graafian follicles that is striking. So often is this the case that one grows skeptical of these terms. A careful examination of true cases of hydrops folliculi shows that neither the ovum nor the membrana granulosa have suffered any appreciable change and I am firmly convinced that the great majority of these ova mature or are cast off by premature rupture of the follicle. In none of these follicles does one find the fatty degeneration or secretion in the tunica interna—a condition which forecasts death of the ovum.

Pfannenstiel says they are usually single and one-chambered, i.e., they do not build daughter cysts. Why would they be single if such cysts were of graafian follicle origin? One would imagine them multiple by the very fact of their origin. Olshausen says when they are multiple, which is seldom, the number is usually small. Most textbooks state that the cysts lie side by side and do not communicate, and the one does not develop out of the other. These arguments seem to have been transcended from textbook to textbook. These two statements so simple in appearance would be among the very hardest possible to prove.

Another arbitrary division is found very frequently, namely, that these cysts are always of limited size; for some the fist, for others the fœtal head is the limit. If they exceed this size, they pass into another category. This seems the most arbitrary of arbitrary divisions. Now it is a singular thing that in the many ovaries that have been examined in the past four years from all the species of animals mentioned in the foregoing pages, never has there been a tumor found arising from the graafian follicles. Hydrops folliculi has been found frequently, but the membrana granulosa was intact. When this had been discharged and had mixed with the liquor folliculi, the cavity became merely a cavity lined by flattened cells of the tunica interna. The small cystic ovary of the dog and cat, and many other animals, has quite another origin, as will be described later.

No one will deny the clinical entity known as "hydrops folliculi," but the interpretation admits of more than one opinion. Nagel believes that these are nothing more or less than unusually large healthy graafian follicles, and I fully concur. We would hardly classify hydramnios as a new-growth, nor does the fœtus succumb even when the quantity of fluid is exceptionally great. Why then should the ovum be doomed under somewhat similar circumstances? The fact remains that hydrops folliculi, when it exists, is nearly always multiple, oftentimes classified clinically as the small cystic ovary, yet according to textbooks, the tumor to which hydrops folliculi is supposed to give origin is in the vast majority of cases single, not multiple.

To my mind there is not one case in the literature of a graafian follicle that is supposed to show developing papilloma, that will stand the test of a scientific criticism. Nagel holds, and I think with perfect justice, that the transformation of the membrana granulosa into a cyst epithelium owing to the death of the eggs, is an impossibility.

In many of the animals in which large and small cysts abound, there are many that can with the greatest ease be traced to their origin, and the same applies to my specimens of human ovaries. Yet in not one of these is there any evidence that cysts, as new-growths, have arisen from graafian follicles. It may be objected that the fact that they have not been found in these series is not proof positive that they do not occur. The whole question seems to resolve itself into this: Are we to consider "hydrops folliculi" as a type of new-growth, or as the product of local change? The question is very difficult. That hydrops folliculi as a clinical entity does exist, no one will deny, but that it can reach anything like large dimensions, I think few will be prepared to admit. Through the work of Kroemer we know that the association of cysts of the ovary with chorio-epithelioma of the uterus is quite a common one. We also know that in the ovaries of a pregnant woman, especially if she is somewhat advanced in her pregnancy, we find not only a large corpus luteum of pregnancy, but the other graafian follicles which contain ova in process of maturation develop a thick layer of lutein cells in their theca interna.

It is as though the fœtal metabolism demanded greater lutein secretion and the theca of these incompletely developed follicles were called on prematurely to do their share of the work. This is the usual course of events as it has been worked out by Seitz and myself.

Such follicles, as Limon has pointed out, the theca interna of which has undergone, or is undergoing this lutein change, have doomed the ova that they contain to destruction.

Chorio-epithelioma acting in a sense like a pregnancy and being derived from a pregnancy, causes all the changes in the ovary that are usually found in a normal pregnancy only to a much greater degree. The ovary becomes markedly cystic and often of considerable volume. If we examine these cysts, we find that they are mostly, if not all, developed from the corpus lutein of pregnancy and from the graafian follicles which were maturing when the abnormal impulse overtook them. The truth of the statement lies in the fact that if these cysts are examined in the early stage they are surrounded by layers of lutein cells just as are those graafian follicles in partial maturation during pregnancy. An impulse to cyst formation seems to sweep over the ovaries in cases of chorio-epithelioma. These are true graafian follicle cysts and not infrequently reach considerable size. Yet, and this is the interesting fact, the large cysts are all devoid of an epithelial lining, being covered either with blood, or serum, or fibrin, or a layer of lutein cells, and only in the smallest can remains of the membrana granulosa be found.

It is very apparent from such cases that a transformation of the lining epithelium of a graafian follicle into the usual lining of a new-growth is an impossibility; that after the death of the ovum the membrana granulosa—a membrane of highly specialized function—has no longer a *raison d'être* and slowly liquefies. If such were not the case, why would not the larger cysts in chorio-epitheliomatous cases still be lined by a membrana granulosa transformed into a one or two layered epithelium?

THE FOLLICULAR CELLS OF THE PRIMORDIAL CELL NESTS

Do epithelial tumors arise from these cells? Theoretically these cells are so closely allied to the germinal epithelium, they have undergone so little differentiation, that there is no apparent reason why theoretically they should not give rise to new-growths. Yet practically such has never been satisfactorily demonstrated. Many authors have published reports of cases in which tumors have been described as having arisen from this structure. The question is a most difficult one to decide, and after all it is a matter of small importance as it will be shown later that these tumors arise either from this primordial follicular epithelium or from invaginations of the germinal epithelium.

Tumors arising out of the follicle cells of primordial follicles have been described by Gottschalk, Steffeck, Bulins, Pozzi and Beausseriat, Pfannenstiel, von Velets, von Kahlden, Hofmeier, Franque and Limnell, none of which, however, seem conclusive, except perhaps that of Gottschalk. Most of the others, as Waldeyer and Heinrichs state, offer other explanations that are much more readily acceptable.

As stated above, even in the bitch where there is normally an invasion of the superficial layer of the ovary by small ducts, which practically never contain normal ova, it is impossible to say under such circumstances whether a tumor arising in this neighborhood, originated in one of these small downgrowths or in a real primordial follicle, and so it is in the human ovum. Small cysts in the immediate neighborhood of the surface epithelium, may so mimic the epithelium of the primordial follicle, that it would be quite impossible to state even at this early stage with any degree of assurance that it arose from the one or the other of these two sources.

THE ORIGIN OF EPITHELIAL TUMORS FROM THE GERMINAL EPITHELIUM

Nothing in the human body can equal the metaplastic power of the germinal epithelium. When we stop to realize that all the epithelial tumors of the ovary (except those few which are classified as inclusion

tumors) are derived from this epithelium either directly or indirectly through the medium of structures to which it primarily gave rise, we are struck with the latent power of these cells when they are awakened into activity. When we realize that such widely different cells as the apparently simple interstitial cells of the ovary at one end of the list, lead, through many mutations, through the follicular epithelium, membrana granulosa, ciliated epithelium mucous cells to the most highly specialized cell—the ovum—we do cease to wonder at the multiplicity of varieties found among the ovarian new-growths.

We have seen in the first half of this monograph, how by a study of comparative anatomy the whole problem of development and atrophy in the human ovary has taken on a new aspect. In that part of the work, I stated that the amount of atrophy of epithelial structures in the late months of fœtal life and early postnatal life varied greatly not only between different species but between different members of the same species. As examples I quoted that the difference between the bitch's and the sheep's ovaries in the matter of the amount of apparently functionless structures retained was very great, but that there were also great differences between any two bitches' or any two of any other species. So it is with the human ovary. In some, even in serial sections, I have found not a trace of any fœtal rests except a small rete ovarii. In others, on the other hand, it is impossible to examine any one preparation without finding not only traces but long stretches of fœtal remnants. This applies equally to ovaries of all ages. What the percentage of cases would be in which such structures are found cannot be stated except by serial sections of a very large number of ovaries at the early adult life. This I have not done, but judging from the frequency with which these structures are found in the ovary before puberty, that is before the ovary is scarred by the rupture of large graafian follicles, the percentage of cases in which fœtal rests occur must be inordinately high—very much above the figures given by Schickele.

These remnants usually lie deep down in the medulla of the ovary, often in the neighborhood of the rete ovarii. At times they are in the form of tubules with well preserved epithelium, at other times the epithelium has remained arrested in a state of partial atrophy. Not infrequently there is a cavity, more frequently they are solid. Often the remains run off from the rete, merely resembling dark lymph spaces. Toward the surface of the ovary one recognizes these fœtal remnants in the deep crevices or lobulations of the surface of the organ, or by the presence of solid columns of cells, or real ducts which communicate with the surface. If these latter occur before fœtal life, yes even before full adolescence, they are undoubtedly of fœtal origin, for none of the causes which could give rise to such sinkings have as yet begun to operate.

There are other fœtal remnants that require description. One frequently finds in human ovaries of all ages certain sectors of the ovary which stand out in marked contrast to the remaining portion of the ovary. Some of these cases will be described. The segment so characteristically changed presents all the appearances of having been arrested in its development, whereby the fœtal structures which should have been absorbed have crystallized and the further development of the segment has not taken place. It is, therefore, a segment which has remained fixed in a stage of imperfect development. A close survey of these cases nearly always leads to a vascular defect as an underlying cause. They present an appearance not unlike what might be the late stage of an infarct. The incidence of these cases in those that have come under my observation shows conclusively that this is a common defect. Let me describe some notable cases:

CASE 1. A fœtus which died at full term. The right ovary normal. The left ovary is normal except for a segment about the middle with apex to the medulla. The surface of the ovary at this spot circular and about 1 centimeter in diameter, is whiter and of firmer consistence. On macroscopic section no change visible except pin-head cavities. Microscopically, a wedge-shaped portion of the ovary, well defined, is devoid of normal ova or its adjuncts. Instead the segment is full of glandular or epithelial elements, some of which are typically

glandular, others are solid columns which at times degenerate into mere lymph-like spaces. The picture is undoubtedly one of arrested atrophy. Other portions of the ovary are quite normal.

CASE 2. A child of one year. Right ovary normal; left ovary contains a segment in the inner pole very similar to that described above. The rest of the ovary is normal.

CASE 3. Girl of 14 years of age. Left ovary normal; right ovary contains at inner pole a wedge of tissue in arrested development. This wedge is filled with epithelial elements and glands. These are frequently broken into short segments. The limits of this wedge are abrupt as if due to an infarct. In many sections the glands contain goblet cells and ciliated epithelium (?).

CASE 4. In this case of a young child of 2 years an irregular segment of the right ovary seems to have suffered a blight. All about this area the normal structures are found whereas in this area which from its outline gives one the impression that it is due to some vascular disturbance arrest of development has taken place. The arborescent fœtal structures are lined with cells that stain a deep blue in marked contrast to the pink and blue of the normal tissues.

I think that such a condition whatever its cause offers an explanation for those rare cases where the ovary remains in its fœtal state, disseminated with canals lined by germinal epithelium, a total absence of ova, a generalized firm fibrous consistence, and a proneness to develop multiple cysts. Frommel (?) has published such a case, and one came under my observation some years ago, when working in the laboratories of the Pathological Institute of Freiburg, in Professor Aschoff's service. Quite recently, a second case, somewhat similar came into my possession. This ovary differing somewhat from any heretofore described, throws so much light upon the origin of tumors that it will repay a thorough study.

It was removed from a young woman of 23 years, married 3½ years, no children. Menstruation late in onset had always been scanty. Uterine dysmenorrhœa was not a marked feature. The external genitals were healthy. Owing to severe left-sided pain of a burning character, which gave all the indications of being due to sigmoidal trouble, but which resisted all forms of palliative treatment, an exploratory laparotomy was done. The sigmoid presented nothing pathological on its peritoneal surface.

The left ovary was slightly, very slightly enlarged, but was hard, and almost cartilaginous in consistence. A few small cysts were visible upon the surface. It was deemed advisable to remove this as being possibly a contributing cause.

Microscopically in none of the sections is there an ovum, either in the quiescent or maturing state. The surface epithelium is unusually tall and columnar. In one of the creases of the surface doubtful cilia are visible. The fibrous stroma is unusually dense and hyaline. The whole ovary is disseminated with small cysts which by their general arrangement run in lines from the surface toward the center of the ovary. Many of these communicated with the surface epithelium by a solid strand of cells. In others this was lacking, but by the course of the fibrous tissue, one could easily see that previously such a connection had existed. The cysts are particularly interesting owing to the variety of their epithelial linings, and the differences in the amount of vascularity of the surrounding fibrous tissue. There are small and large cysts (microscopically); cysts without any apparent epithelial lining, others with a flat lymph space-like covering; still others with cuboidal cells. In this cyst we see tall cylindrical cells, in that one goblet cells, and still further on, undoubted ciliated epithelium. In one of the larger cysts the wall has become very vascular and papillary ingrowths are quite frequent.

That this is an ovary arrested in its embryonic development, I think cannot be doubted. It presents all the features of such a condition. How such a condition can arise is indeed quite another problem. And yet I possess among my specimens two which I prize most highly. These were obtained during my work at the Friedrichsheim Krankenhaus in Berlin. The first was that of a child of five years which had died of chronic nephritis, secondary to scarlet fever. The one ovary was quite normal in every respect. The second was much smaller. The characters of the latter, the evident atrophy of the parenchyma, the shrinkage of the tissues, led me to examine the vessels in the hilus. These were unusually small and cross sections of the main trunks of the vessels farther out in the broad ligament and of the ovarian and uterine vessels of that side showed almost complete closure due either to a developmental defect or to an early obliterative endarteritis.

The second case was that of a girl of eighteen, who died of brain tumor of syphilitic origin. One ovary was perfectly healthy, ovulation had gone on, and there were slight remains of lutein cells and of corpora throughout the ovary. Microscopically this was a perfectly normal ovary. The other ovary was much smaller. The contrast under the micro-

scope could not be more marked. This ovary was like that from a woman of 50 years. The fibrous tissue was seemingly more adult in type. There were a few ova which had begun to mature. The most of these never reached an advanced state of development. The ovary therefore contained retention cavities, the results of these atrophic graafian follicles. But the most striking feature was the presence of many corpora candicantia, which are similar to those found in the aged who have not sufficient vitality totally to destroy and totally to absorb. This artery was also the seat of a chronic obliterating syphilitic endarteritis.

I have quoted this last case as showing the effect of faulty vascularization in the adult, and as throwing some light perhaps upon the previously described cases, which occurred during the developmental or embryonic state, when any change in the blood supply would lead to faulty destruction and absorption of redundant tissues.

Of course the only effect such change could have on the adult ovary would be to retard destruction and absorption of functional vessel scleroses and absorption of the products of corpora lutea and corpora atretica thereby causing such an ovary to resemble that of an aged woman.

Pfannenstiel has described cases in which the surface epithelium bore patches of ciliated epithelium. Some of these ovaries were quite normal. Others were the seat of tumor growths. Desinety also found islands of ciliated epithelium upon the surface of otherwise normal ovaries. Waldeyer found it in six different cases, in five of which there was cystic formation, and the sixth was the seat of chronic inflammation. Marchand to explain its presence upon the surface of the ovary, stated that it arose from the fimbria ovarica, and became detached from it. Williams, Walthard, and Kuszman, as previously stated, claimed also that these were isolated portions of the muellerian duct. In one of my specimens, there are islands of ciliated epithelium in a depression on the surface of the ovary.

Today it is fully recognized that there is a close relationship between the germinal epithelium and ciliated epithelium, and that the transition from the one type to the other is frequent and easy, and seems but the expression of a character which germinal epithelium once permanently possessed. Pick has drawn attention to the general tendency of the whole of the pelvic peritoneal surface to develop cilia.

In addition to the foregoing cases, which so far as it is possible to ascertain, date back their origin to fœtal life, there are other characteristic invasions of the ovarian stroma by the germinal epithelium, which leave no doubt but that they are acquired conditions, due to age changes and to inflammatory changes. Inflammation of the pelvic peritoneum giving rise to chronic perioophoritis, is a fruitful source of cyst formation. Clinical experience teaches, I think, that new-growths are not commonly associated or secondary to inflammatory changes. These inflammatory cysts usually subside with the subsidence of the inflammation. One can readily conceive that inflammation about an ovary containing many fœtal remnants may act as the stimulus that starts these into activity. In such cases the inflammation would therefore be merely an accidental association.

TUMORS ARISING FROM OVULES

The dermoids and teratomata. This constitutes perhaps the most interesting chapter of all those dealing with the subject of tumor formations in the ovary, and of late so much has been added to our knowledge concerning their origin, that today there is but little doubt that the ovules by a system of pathenogenesis are responsible for the presence of dermoids and teratomata. The evolution of this idea is interesting and facts that really bear upon the subject are indeed of but very recent date. Previous to this the whole subject lay in the domain of theory, and there are none that are more productive of controversy than just such.

The earliest theory held that teratomata and dermoids were of the nature of mixed tumors, arising from fœtal rests of already differentiated cells in the ovary. It also held that these were not normally developed in the ovary, but were due to a misplacement from other organs. Of late, this theory was again brought to light by Bandler who contended not only that this theory was well founded, but that the pronephros and the

wolffian body were responsible for these inclusions. In the light of our present knowledge, we know that not only the theory is wrong, but also that there was a misinterpretation of the facts from which the theory was developed.

Later Wilms pointed out by a careful examination of dermoids and teratomata, that the former were not the simple mixed tumors as generally held, but that they were mixed in the sense that they might contain structures developed from all the three primitive layers of the fœtus, but that the ectodermic layer or ectoblast usually was so predominately developed as to mask the other two, whereas in teratomata, it was usually the mesoblast which gained the ascendency. In other words, both these tumor types were really of only one kind for they both contained products of the three fœtal germinal layers, namely, the ectoblast, mesoblast, and endoblast. The difference, therefore, was one of relative quantities of these and not of quality.

But of late, as if stimulated by the warmth of the discussion between these two contending camps, the subject of parthenogenesis has attracted a great deal of earnest work. Within the last twelve years we find Delage and Bataillon busy over this subject in France. Loeb, Wolfsohn, Morgan and Weir in America, and later there are added Wedekind, Daudin, Arbacia and Kostanechi. The sum and substance of their work has led to a great deal of weight being added to Pfannenstiel's theory. Through the experimental work of these, it has been established beyond a doubt that parthenogenesis can be provoked experimentally, in echinoderms, worms, molluscs, anthropodes, fish and amphibians. Natural parthenogenesis has also been observed in mammals by Bischoff and Hensen, in batraciens by Bischoff, Leuckart, Born and Dehner, and in fish, worms, anthropodes, echinoderms and molluscs by a number of authors.

Lecaillon who has done a great deal of work upon this subject states: "We can consider as proved beyond all possibility of doubt that parthenogenesis really takes place naturally in many animals that are of widely different species. Parthenogenesis is really evoked by this fact, and this fact alone, that the unfertilized ovum is endowed with the ability and property to evolve along the lines of embryonic development of segmentation and differentiation, and not because the egg has encountered special stimulus and special surroundings. This is proved by the fact that in birds the segmentation of non-impregnated ovum takes place as the egg passes along the oviduct. Such segmentation therefore takes place under the same circumstances and surroundings as it does with the impregnated ovum which segments normally.

Pfannenstiel had stated that no dermoid or teratoma has been found sufficiently early to enable an opinion upon its origin. That was reserved for Loeb.

One year later the author found that parthenogenesis is a common occurrence in the guinea-pig, and that the ovum is the starting point of this development. He found that parthenogenesis occurs in about 10 per cent of guinea-pigs before they are 6 months old. Later, the condition is much less frequent. When describing one of these cases he writes: "We see in each case a choriomic vesicle with trophoblast and plasmodia and syncytia penetrating into the neighboring tissue. There is also a structure present which is probably to be interpreted as the neural tube. As this type of growth occurs in the cortex of the ovary where follicles are normally seen, and are found within follicle-like cavities, they can be derived only from ova developing parthenogenetically. Fertilization can be excluded as the life history of some of these animals is known and precludes such interpretation. It is very probable that parthenogenetic change sets in soon after ovulation, the altered conditions in the ovaries at that time (variations in blood pressure, in intrafollicular pressure, changes in oxygen supply) supply the necessary stimulus.

The later stages of these developing embryo bear some resemblance to chorio-epitheliomata. These observations demonstrate that chorio-epitheliomata and teratoid tumors that are found in the ovaries are not derived from misplaced blastomeres, as Bon-

net and Marchand believed, but that the older view is correct according to which at least the teratoid tumors of the ovary are derived from parthenogenetically developed ova.

Loeb might have gone one step further and included the dermoids, for histogenetically they are the same as shown above.

As for those extragenital cases, there seems as yet no adequate scientific explanation forthcoming. Pfannenstiel maintains that those of the trunk and peritoneal cavity have the same origin as those of the ovary. The assumption is that they arise from ectopic ovarian tissue which we know is found not only throughout the pelvis, but throughout the abdominal cavity, and it is well-known that the seat of the vast majority of these extragenital dermoids and teratomata are localized in the pelvis. Nagel, Minot and Ribbert have found wandering ova throughout the genital abdominal cavity. "Grant such a wandering of ova," states Pfannenstiel, "then the propagation of derivative tumors allows of a very ready and easy explanation."

Do pseudomucinous cysts arise from teratomata and therefore from the ova?

The frequent association of this type of cyst with the teratomata is more than accidental. Yet we are struck with the frequency with which the dermoids are surrounded by other cysts whose origin is known to be from the germinal epithelium of the ovary. As stated above Landau, Hanan, Ribbert and Askanazy, contend that the pseudomucinous cyst, whether alone or in association with dermoids is part and parcel of the dermoid and the expression of the development of the intestinal portion of the teratoma or dermoid just as the teeth, brain and jaw, and hair, are the expression of its cephalic portion.

If we look into the question more closely, we find that not only do pseudomucinous cysts surround dermoids, but the incidence of simple serous cysts in association with dermoids is more than a casual association. It would seem that dermoids by their irritation and growth stimulate the surrounding tissues to activity. We have seen how the vast majority of human ovaries contain all the necessary epithelial rests or late invasions for the development of cysts and tumors. We have seen how these rests or invasions, derived as they are from the germinal epithelium, possess the wonderful metaplastic properties of this epithelium. We have even seen how in certain animals these rests can produce ova and pseudo-ova, none of which, however, perhaps ever reach maturity, and lastly I have shown how the germinal epithelium can and does transform itself into mucous cells that have all the characters of the lining cells of the pseudomucinous cyst.

I have found several of my cases of dermoids with mucin mixed with the sebaceous material. Microscopic section always shows in corroboration of the microscopic description that this is due not to a portion of the original wall of the dermoid being a mucous surface as you would expect to find if the pseudomucinous cyst were part and parcel of the dermoid, but in each case it was quite apparent that the surface covered with mucous cells had once been a cyst separate and apart, and had later been incorporated. This fact taken with the many stated in this chapter cannot but lead, I think, to the conviction that pseudomucinous cysts are not only ovulogenic but are also of germinal epithelium origin.

The interstitial cells of the ovary. Upon the subject of tumors arising from this tissue, nothing so far has appeared in the literature. Inasmuch as this monograph deals with epithelial tumors of the ovary, and since it has been shown that the interstitial cells of the ovary are of epithelial origin, this tissue must be taken into consideration as a possible factor in the production of epithelial tumors.

It is singular that in many of the so-called peritheliomata the tissues change almost imperceptibly into carcinomata on the one hand and into alveolar sarcomata on the other hand. Particularly is this true of peritheliomata of the mucous membrane of the intestine. More especially are such tumor-growths found in the appendix vermiformis. I have seen several such in which it was quite impossible to state whether the new-

growth was of the nature of a carcinoma, or of a perithelioma or of a sarcoma, for all three types existed in the same specimen. There is a striking fact in connection with the ovary. In our series of malignant diseases of the ovaries at the Royal Victoria Hospital—a series extending over ten years—peritheliomata are eight times more frequent than are the sarcomata. In fact sarcoma of the ovary is in my experience a rare tumor of the ovary. This observation is confirmed by reports from the literature.

In view of these facts, I think the theory quite justified, that inasmuch as peritheliomata occupy morphologically a position midway between carcinomata and sarcomata, its relative frequency in the ovary might readily be explained by assuming that it arises from the interstitial cells which occupy a position morphologically between the epithelial and connective-tissue cells. The incidence of peritheliomata of the ovary in the ratio of eight to one of sarcoma has something more in it than will be accounted for by the law of accidental occurrence. Such a preponderance of peritheliomata in the ovary is found in no other organ in the human body. In fact the ratio is usually eight sarcomata to one peritheliomata. To me it is quite doubtful whether such a cell as the interstitial cell, could completely abdicate its primitive characteristics and so deny its origin as to develop into a sarcoma. We must not look upon the interstitial cell as being of a nature of a connective-tissue cell. It has a much more exalted function. We have seen how periodically when in the neighborhood of maturing graafian follicles it can awaken into activity and assume secretory functions and serve very materially in the maintenance of equilibrium in the internal economy. Its position morphologically speaking between the connective tissue and the epithelial structures make it preeminently the tissue responsible for the great frequency of perithelioma in the ovary.

PUERPERAL INFECTION; A PLEA FOR EARLY OPERATION IN PELVIC SEPTIC PHLEBITIS

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GENERAL INTRODUCTION

OFTEN masquerading under strange titles, puerperal infection is fraught with much tragedy for humanity. In English speaking countries alone it claims at least 10,000 victims every year. This estimate is necessarily higher than that of official statistics, since in British communities the fatal issue is often ascribed to such diseases as appendicitis, peritonitis, enteric fever, while Professor Whitridge Williams states that in America the majority of deaths from puerperal infection are set down to other causes.

The erroneous causes of death so frequently alleged in puerperal infection may represent mistakes in diagnosis but are often due to the medical attendant realizing that a frank admission of puerperal infection may leave him open to the charge of having "left something behind," or of having been unclean in his work. There may be no foundation for such charges, all aseptic care having been used, and not a particle of placenta being retained; on the other hand, the most scrupulously careful examination may fail to discover whether a freshly delivered placenta is absolutely complete.

In order to appreciate the full significance of the foregoing statements, it will be necessary to make some reference to the disease which Virchow named *polypoid decidual endometritis*, and which I discussed at length in 1918 in the March number of Surgery Gynecology and Obstetrics.

POLYPOID DECIDUAL ENDOMETRITIS

In the majority of the hundreds of cases of puerperal infection admitted to the Perth Hospital I have found polypoid decidual endometritis, with or without adherent placenta; so that the commonly assumed rarity of this disease of the decidua would appear to lie not in its occurrence but in its recognition. In works on puerperal infection and on obstetrics excellent examples of the disease are figured as "hypertrophied decidua," "necrotic decidua," "roughened placental site," and so forth. As time goes on these misleading titles will doubtless give place to the correct one "polypoid decidual endometritis," as with Professor Sir Harry Allen's specimens in the Melbourne University.

Polypoid decidual endometritis was mentioned first by Virchow in 1861 in connection with a three months' abortion, in which he found polypoid outgrowths of the decidua suggestive of condylomata. Following on Virchow's single observation, others as Gusserow, Bulius, Strassmann, and Ahlfeld described odd cases in abortions. Later (1908) Frank Nyulasy, of Melbourne, showed that the disease is a relatively common (3 to 5 per cent) complication of pregnancy, and is not seldom met with after full-time labor, a fact of high importance, since, although the placenta may be expelled complete, the patient may nevertheless die of puerperal infection dependent on the polypoid decidua. He worked out a satisfactory minute pathology, symptomatology, and treatment, and proved that the great outstanding feature of the disease is that the involved decidua is signally vulnerable to infection.

According to Frank Nyulasy, polypoid decidual endometritis is an interstitial inflammation of the decidua and partly of the underlying muscle. Depending on the extent and distribution of the interstitial fibrous tissue, the affected decidua may take the form of tough polypoid or papillomatous outgrowths, or may be merely tough, thickened, and uneven. The disease is a common cause of abortion and of adherent placenta.

PELVIC SEPTIC PHLEBITIS

Frequency and clinical types. The majority of fatal cases of puerperal infection fundamentally associated with the interior

of the uterus have, in my experience, shown pelvic septic phlebitis. On this point Lea states that this complication is responsible for from 30 to 50 per cent of all deaths from puerperal infection. The importance, then, of any surgical measure calculated to diminish the mortality from pelvic septic phlebitis can hardly be overemphasized. But while accepting this view, the surgeon is often confronted with the difficulty of discovering definite indications for operation. In cases with recurring rigors, in which the thrombosed veins can be palpated, there need usually be little hesitation, and brilliant results have been achieved by excision or ligature of the infected veins in a limited number of cases described in the literature. Such clear-cut cases of thrombophlebitis are, I believe, the exception, and we more often have cases admitted to hospital in which there is merely a subinvoluted uterus, but without any discoverable extra-uterine lesion, and in which there may be no rigors. With the latter group this article is mainly concerned, as they are the cases which, hitherto at least, have only too often been the despair of the surgeon. In what follows I hope to indicate rational lines for their early recognition, with a view to prompt operation, since delay may mean death. The following case is an example of surgery having been invoked too late.

CASE—GROUP A; LATE OPERATION

This case was admitted several days after labor with the symptoms of puerperal septicæmia. Examination revealed a subinvoluted uterus and extensive polypoid decidual endometritis, with some adherent placenta. In spite of clearing out the uterus and other treatment, the patient continued to deteriorate. When at this time I left for a holiday no gross extra-uterine lesion could be palpated, and no rigors had occurred. On my return in about 7 weeks, the patient was in a grave condition, and having rigors daily. Enlarged tubes were now detected, and on at once opening the abdomen (May 20, 1917) extensively thrombosed spermatic veins and double pyosalpinx were found. The thrombosed veins were excised to as high a level as possible, and the appendages removed. Pus was present in the left vein. After operation there were no more rigors, but in spite of some temporary improvement, the patient died in 8 days.

Importance of early operation. The temporary improvement after excision of infected veins noted in cases such as the foregoing suggested that in sufficiently early interference might be found a key which would open the door to new surgical triumphs. I realized that a proportion of cases terminate in natural recovery, but I was satisfied also that the majority (Lea says 60 per cent) terminate in death. It seemed to me then that the surgeon's duty to operate early should be even more imperative than in appendicitis, in which most of the cases are very properly submitted to early operation, although the majority would not end fatally in the absence of surgical intervention.

Diagnosis. In a patient with puerperal infection fundamentally connected with the interior of the uterus, as is most commonly the case, the relative frequency of polypoid decidual endometritis as a complication of pregnancy, and the marked vulnerability of the involved decidua to infection should be clearly borne in mind. Subinvolution, which is always a feature of polypoid decidual endometritis, indicates urgent need for examination of the interior of the uterus. If polypoid decidua (and any adherent placenta) is removed in the early stage, when merely subinvolution is present without perhaps quick pulse, fever, hæmorrhage, or foul discharge, the patient is practically certain to recover; otherwise, by delay she may die of puerperal infection in spite of late removal of the polypoid decidua.

Let us assume that in a case of puerperal infection, the uterus is cleared of any polypoid decidua and of any adherent placenta, and that examination has failed to discover any gross extra-uterine lesion in the pelvis or elsewhere. Thence onward it becomes a question of intelligent after-treatment and careful observation, the pulse respirations and temperature being taken every 2 hours, and in graver cases every hour. It is only by such short-interval records that we can gauge the progress of the case with any real accuracy.

If after some days—and no more definite rule can at present be formulated—the patient continues to deteriorate from puerperal infection, the diagnosis of pelvic septic phlebitis becomes largely a matter of exclu-

Fig. 1. Perth Hospital specimen: vertical section through uterus showing polypoid decidua. Patient admitted a week after labor with septicæmia, suppurative peritonitis, ovarian veins thrombosed, double pyosalpinx.

Fig. 2. Photomicrograph of section through polypoid decidua in Fig.1, showing enormously dilated capillaries and interstitial fibrous tissue. Other sections showed marked endarteritis obliterans, but no glands discovered. Both sections and photos were made at Melbourne University through the courtesy of Prof. Sir Harry Allen.

sion. If no other pelvic lesion can be recognized, and no other septic focus (uterine or otherwise) can be discovered, we are, I think, generally justified in assuming the existence of pelvic septic phlebitis. The presence of localizing pain, or of rigors, or of femoral phlebitis would strengthen our diagnosis; the absence of these signs has no probative value either way. In the cases I have seen the thrombosed veins could not as a rule be palpated, so that in such circumstances the progressive deterioration is the only really safe indication to follow. Having then provisionally diagnosed thrombophlebitis, operation should be carried out without delay.

In cases such as that just outlined, might not the septic symptoms be dependent entirely on extensive and virulent infection of the uterine substance? Assuming the uterus has been cleared out, the only sign that would definitely suggest the condition would be marked tenderness of the uterus to pressure. Infection of the uterus of so serious a character, without thrombophlebitis, has seldom come within my experience, although Henry Jellett successfully tied the ovarian veins in one such case, the supply of poison from the infected uterus being in this way shut off. In a similar case in which I tied the ovarian veins some years ago, the patient quickly died of acute œdema of the lungs caused by six pints of saline intravenously injected by a too zealous assistant.

If in infection of the uterine substance alone there was evidence on cœliotomy of the infectious process leading to perforation, as by areas of softening or actual solution of continuity, hysterectomy with wide excision of the ovarian veins should be carried out.

The operations performed for thrombophlebitis are most commonly excision of the ovarian veins, and less frequently ligature of the internal iliac. In very extensive thrombosis the common iliac has been successfully tied, and Warneckros suggests ligature even of the inferior vena cava.

The case now to be described fairly well represents the practical application of the views herein enunciated. In five other cases,

pyosalpinx of greater or lesser degree was present with thrombosis of the ovarian veins, four of these cases recovering by operation.

CASE—GROUP B; EARLY OPERATION

A woman, aged 26, was sent in for "appendicitis" about ten days after labor in a public maternity hospital. She complained of pain in the right iliac region, but no tumor could be palpated, and the temperature chart made before admission was of a distinctly septic type. The pulse was 120, and the temperature raised, while the uterus was large and soft. There were no rigors before or subsequent to admission. Under anæsthesia much polypoid decidua was removed, only one very small particle of placenta being detected. The patient improved to such an extent that after a day or two her general condition ceased to give cause for serious anxiety while the iliac pain was relieved by local applications. At the end of about a fortnight, however, during the course of a day or two, she showed somewhat rapidly signs of marked deterioration, her color becoming bad, the pulse reaching 136, respirations 35, and temperature 103° F., while the iliac pain was again obtrusive. Up until this time the pulse had varied from 80 to 100, and the respirations and temperature were not much over the normal. No gross pathology could be palpated in the pelvis, the uterus was not tender, and no septic focus could be discovered elsewhere. On opening the abdomen (July 6, 1918) the right ovarian vein was found to be thrombosed, and adjacent intestines acutely inflamed and adherent; the uterine appendages were normal, while the appendix was healthy and almost entirely retrocæcal. The thrombosed vein was removed, and the abdomen closed without drainage. The pain very soon disappeared, the general condition quickly improved, with sharply falling pulse and respiration rates and the patient was discharged after a few weeks convalescence in the open air.

GENERAL CONCLUSIONS

The recovery of cases such as that just described encourages me to believe that early operation may save many cases of puerperal infection which would be lost when treated on the lines hitherto followed. Indeed, I have now come to consider it absolutely mandatory to open the abdomen in cases of puerperal infection, the moment there are the grounds, which I have indicated, for the diagnosis of pelvic septic phlebitis, and apart from any gross pelvic lesions discoverable by the known methods of physical diagnosis, and always assuming that the interior of the uterus has been cleared out. To me early operation in such cases has become a supreme duty.

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A UROLOGIC AND RADIOGRAPHIC STUDY OF THE SAMAR TWINS

With a View to Surgical Separation of the Bodies

By H. W. PLAGGEMEYER, M.D., F.A.C.S., Detroit, Michigan; and J. H. SELBY, M.D., Washington

THE Samar twins, Lucio and Simplicio Godino, were studied by us, at the request of the Brooklyn Society for the Prevention of Cruelty to Children, with a view to their complete separation by surgical intervention and as to the extent of possible danger attending such procedure. To this end, a careful observation of their gastro-intestinal tracts through the medium of the X-ray was made, combined with a differential study of the urological tracts, with a consideration of the interrelation, if any, of their blood vascular systems. The work was done at the Walter Reed Hospital.

The results of our observations are briefly stated below. That we were unable to follow our original intention of observing blood changes and excretory phenomena following test meals, as well as of producing a complete set of bladder pictures, was due to the inherent difficulty of getting absolute co-operation on the part of the twins themselves. We deem ourselves extremely fortunate to be able to present the following data.

RADIOGRAPHIC FINDINGS (J. H. SELBY)

The combined fluoroscopic and stereoradiographic examination before and during a barium injection of the colon revealed the following:

1. There is no bony union between any portion of the twins

2. The coccyx of Lucio, the larger twin, is well developed, as compared with that of Simplicio, which is rudimentary in type.

3. Lucio presents a functionating anus, while Simplicio presents an imperforate rectum, represented by a dimple which barely admits the end of the little finger, and is about one-fourth of an inch deep.

Injection of the colon was difficult, in that it was shown that there was no synchronism between the peristaltic action of the two colons. The irritation of the anal sphincter caused by introduction of the enema nozzle was appreciated by Lucio but not by Simplicio. Lucio experienced a slight discomfort, as soon as the enema solution entered his rectum, while Simplicio was not aware of its presence until the solution was seen by the fluoroscope to pass across the mid line into his portion of the joined bowel. From that time on to the expiration of the 30 minutes required for the injection, first one twin and then the other complained of an impending bowel movement. This alternation in their desire for defæcation was an interesting phenomenon, as opposed to the stimulation of urination, which invariably was simultaneous. The bowel injection was finally accomplished so that we were enabled to distinguish the outlines of the anastomosis.

It was found that the colon anastomosis was roughly "H" shaped, the lower left prong of the "H" corresponding to the rectum of Lucio; the lower right prong of the "H" simulated a small diverticulum, projecting caudalward to within about 1 centimeter of the dimple. The upper left prong of the "H" corresponded to the sigmoid of Lucio, and the upper right prong to the sigmoid of Simplicio, the cross bar representing the anastomosis.

At one time during the fluoroscopic observation, the solution was seen to ascend in the sigmoid of Simplicio while it was at a standstill in the sigmoid of Lucio. At this moment Lucio was complaining bitterly of his inability to retain the solution. At another time Lucio was remonstrating with Simplicio for complaining of his discomfort.

UROLOGICAL REPORT (H. W. PLAGGEMEYER)

An examination of the external genitalia of the twins, showed them *each* to possess a normal penis for the pre-puberal eleven-year-old male, with distinct and individual scrotal sacs, each boy having two descended testes, with epididymes and corpora within range of normal.

Fig. 1. The twins in normal standing position. Simplicio is slightly taller and stronger than Lucio. There is, however, a perfectly balanced reciprocal action in all their movements. They run up and down stairs, jump off chairs, etc., in perfect unison, and there is never any apparent indecision in their mutual co-ordination.

From the fact that urination was simultaneous through both urethræ, it was thought the twins might be possessed of a common bladder, and possibly of a common system of kidneys and ureters. To this end I resorted to the following simple method of differentiation.

Renal function. Phenolsulphonephthalein was used. Intravenous injection was not resorted to, owing to most strenuous objections on the part of the twins, and it was with difficulty they were induced to accept the deltoid intramuscular approach. As actual percentages were not desired this was considered satisfactory. Both twins flatly objected to catheterization, even with a No. 8 ureteral catheter, but, as it happened, this also was not necessary.

First day, May 19, 1919: They were each given at 3:30 p.m. 500 cubic centimeters of water to drink, with the following results:

Lucio, injected right deltoid 6 milligrams phenolsulphonephthalein. Injection 4:00 p.m.

		Lucio	Simplicio
Urination (simultaneous):	4:05 p. m.	No dye	No dye
	4:08 p. m.	No dye	No dye
First half hour	4:10 p. m.	Dye present	No dye
	4:14 p. m.	Dye present	No dye
	4:15 p. m.	Dye present	No dye
	4:16 p. m.	Dye present	No dye
	4:18 p. m.	Dye present	No dye
	4:21 p. m.	Dye present	No dye
	4:23 p. m.	Dye present	No dye
	4:29 p. m.	Dye present	No dye
	4:31 p. m.	Dye present	No dye
	4:34 p. m.	Dye present	No dye
	4:36 p. m.	Dye present	No dye
	4:39 p. m.	Dye present	No dye
	4:40 p. m.	Dye present	No dye

Total 30 minutes:
Lucio, 250 cubic centimeters; 23 per cent phenolsulphonephthalein; specific gravity, 1,006.
Simplicio, 210 cubic centimeters; 0 per cent phenolsulphonephthalein; specific gravity, 1,006.

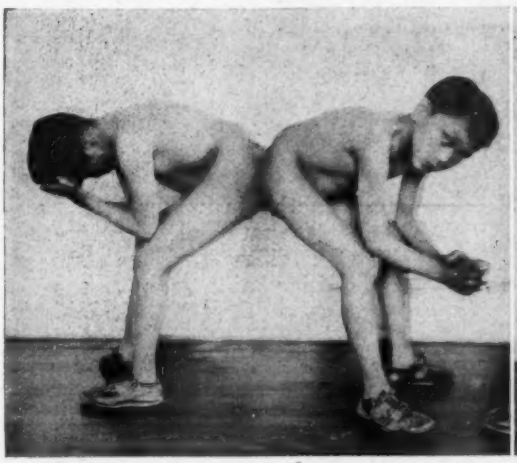
		Lucio	Simplicio
Second half hour	4:40 p. m.	Dye present	No dye
	4:45 p. m.	Dye present	No dye
	4:50 p. m.	Dye present	No dye
	4:52 p. m.	Dye present	No dye
	4:55 p. m.	Dye present	No dye
	4:59 p. m.	Dye present	No dye
	5:01 p. m.	Dye present	No dye
	5:04 p. m.	Dye present	No dye
	5:07 p. m.	Dye present	No dye
	5:10 p. m.	Dye present	No dye

Total 30 minutes:
Lucio, 326 cubic centimeters; 18 per cent phenolsulphonephthalein; specific gravity, 1,007.
Simplicio, 325 cubic centimeters; 0 per cent phenolsulphonephthalein; specific gravity, 1,007.
Lucio, first 30 minutes: phenolsulphonephthalein, 23 per cent; fluid amount, 250 cubic centimeters. Second 30 minutes: 18 per cent; fluid amount, 326 cubic centimeters. Total, 41 per cent, 576 cubic centimeters.
Simplicio, first 30 minutes: phenolsulphonephthalein, 0 per cent; fluid amount, 210 cubic centimeters. Second 30 minutes: 0 per cent; fluid amount, 325 cubic centimeters. Total, 0 per cent, 535 cubic centimeters.

Simplicio was watched for 5 hours, and failed to show any trace of dye. That they might possibly be possessed of one bladder was therefore ruled out when the phenolsulphonephthalein tests were made.

Second day: Simplicio was used as host; 6 milligrams of phenolsulphonephthalein being injected into right deltoid. Both urines were negative to phenolsulphonephthalein before injection. Each was given 500 cubic centimeters of water by mouth at 2:00 p.m. May 20, 1919, Simplicio: injected right deltoid, 6 milligrams phenolsulphonephthalein 2:19 p.m. Appearance time, seven minutes. The day was very warm, excretion by skin more active, and urination not so frequent as on preceding day.

		Lucio	Simplicio
Urination (simultaneous)	2:22 p. m.	Negative	Negative
	2:26 p. m.	Negative	Dye appeared
First half hour	2:27 p. m.	Negative	Dye present
	2:45 p. m.	Negative	Dye present
	2:55 p. m.	Negative	Dye present
	2:59 p. m.	Negative	Dye present



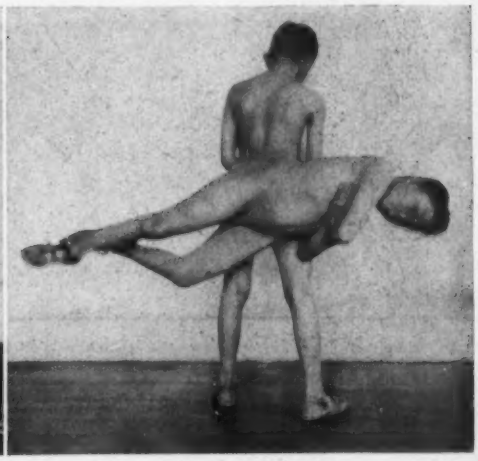


Fig. 2. (at left). There is no evidence of spina bifida. The circumference of connection stalk is 14 inches.

Fig. 3. Lucio is throwing Simplicio in torque. In movements of this type he is usually the more prone to lift his brother, and Simplicio swings himself with ease, due to the rudimentary form of his coccyx.

		Lucio	Simplicio
First half hour volume		350 ccm.	230 ccm.
Function		0 per cent	30 per cent
Urination	3:08 p. m.	No dye	Dye present
Second half hour	3:18 p. m.	No dye	Dye present
	3:30 p. m.	No dye	Dye present
Second half hour volume		260 ccm.	180 ccm.
Function		0 per cent	19 per cent
Urination	3:42 p. m.	No dye	Dye present
Third half hour	3:51 p. m.	No dye	Dye present
	4:00 p. m.	No dye	Dye present
Third half hour volume		275 ccm.	185 ccm.
Function		0 per cent	10 per cent
Urination	4:15 p. m.	No dye	Dye present
Fourth half hour	4:30 p. m.	No dye	Dye present
Fourth half hour volume		220 ccm.	205 ccm.
Function		0 per cent	6 per cent
Total two hours volume		830 ccm.	615 ccm.
Phenolsulphonephthalein		No dye	65 per cent (49+16)
Specific gravity		1.005	1.003

Third day, May 23, 1919: Methylene blue 5 grains given to Lucio by mouth at 12:40 p.m. At this time specific gravities were: Lucio, 1,009; Simplicio, 1,023. Lucio was given 500 cubic centimeters water by mouth at 12:40 p.m.

		Lucio	Simplicio
Urination	2:20 p. m.	Blue	Clear
	2:40 p. m.	Blue	Clear
	2:50 p. m.	Blue	Clear
	3:05 p. m.	Blue	Clear
	3:35 p. m.	Blue	Clear
	3:40 p. m.	Blue	Clear
	3:45 p. m.	Blue	Clear
	4:05 p. m.	Blue	Clear
	4:30 p. m.	Blue (1,005)	Tr. blue (1,024)

Lucio had been given copious water, Simplicio none. At 4:30 specific gravities were: Lucio, 1,005; Simplicio, 1,024. Amounts: Lucio, 655 cubic centimeters; Simplicio, 115 cubic centimeters.

Seventh day, May 27, 1919: Methylene blue given Simplicio, at 10:00 a.m. At 3:00 p.m. Simplicio very dark blue; specific gravity, 1,007; Lucio, pale blue; specific gravity, 1,015.

May 28, 1919: Lucio was given 10 grains methylene blue by mouth at 10:00 a. m. It appeared in the urine in 1 hour and 30 minutes in both, though Simplicio had been given no water. Specific gravity: Lucio, 1,013; Simplicio, 1,022.

SUMMARY OF UROLOGICAL FINDINGS

Phenolsulphonephthalein injected in deltoid of each boy alternately, fails to materialize in the other within five hours, the period of observation.

Methylene blue, 5 grains by mouth to each alternately as host appears in the other urine at varying periods following appearance in the host, but the concentration of the dye is always greater in the host, even, though, under forced hydration his specific gravity is absolutely and relatively lowered.

Specific gravities can be raised and lowered at will in each. When one is given water, and the other restrained, specific gravity in the latter remains apparently unaffected by absorption from lower intestine of host.

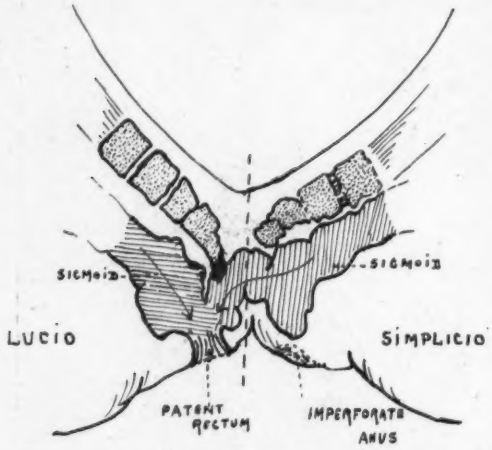


Fig. 4. Conventional diagram of roentgenogram. In the original plate the connecting arm between the two sigmoids is plainly seen, but the clarity of the sigmoidal outline is, unfortunately, lost in the print.

Both bloods are compatible, and classified under Type II (Moss classification).

It is evident from this brief study that the urinary tracts have nothing in common. The bladders and ureters are evidently distinct. The fact that they urinate simultaneously is partly psychic and partly due to the fact that the trigonal portions of the bladders are probably apposed, and the wave-like contractions in one bladder incident to distention stimulation cause muscular and fibrous contractions in one bladder, which reflexly set up a like action in the other. Then, too, there may be an interposition of fibers from the anterior branches of the nervi erigentes. This is doubtful.

The fact that phthalein does not go through, and that the pulse rate and blood pressure were at all times different, shows a lack of common blood circulation. The methylene blue apparently went through in the intrinsic vessels of the sigmoid. This is plausible from the picture in the radiographic plates.

We feel convinced from our observations on these boys, that they can be separated with relative ease, the chief difficulty to be encountered being centered in the attack on the sigmoids, and the correction of imperforate anus on one side. The urological tract should offer no difficulty.

As the boys are now eleven years old, and rapidly approaching the age of puberty, with its attendant changes in the pelvic blood supply, the present is the most auspicious time for surgical approach.

We wish to extend our thanks to Colonel W. L. Kellar, on whose service these observations were made, and to Lieutenant William D. Gill for his care in typing the bloods.

ACUTE OSTEOMYELITIS AND PERIOSTEITIS COMPLICATING EPIDEMIC INFLUENZA

Report of Five Cases; Radius Removed in One Case; a Review of the Literature of Excision of the Radius

By MOSES BEHREND, M.D., Philadelphia

IN this small series of cases of acute osteomyelitis complicating epidemic influenza, three out of five cases occurred in children under ten years of age, the youngest being an infant one year old, the oldest a child of 9. As compared with some of the other complications of influenza, such as pneumonia and empyema, osteomyelitis plays a minor rôle. The period of onset of the symptoms of acute osteomyelitis, varies, the manifestations appearing weeks after the acute symptoms of influenza have subsided. In two of the cases an interval of 5 weeks elapsed before the symptoms of acute osteomyelitis appeared.

One fact has not been fully realized by the profession and that is that dangerous complications result if an acute osteomyelitis is not operated upon. The destruction of tissue and in some instances even the death of the patient, make it just as imperative that operation should be done in such cases as in cases of appendicitis and the measures used should be just as radical. The sooner the medullary canal is opened, the better will be the ultimate functional result. The only safe procedure is the removal of the entire roof of the bone (Fig. 1, Case 5). This insures adequate drainage, thereby enhancing the healing process which, as we all know, is a long process at best.

A search of the recent literature, up to and including January, 1919, discloses no reference to acute osteomyelitis as a complication of influenza. The cases reported in the early literature are few in number. Leclere saw a case of osteomyelitis of the maxilla following influenza. The patient had had an injury to this bone 5 years before. André makes no mention of osteomyelitis as a complication of influenza. Franke reports several cases in which periosteitis and osteomyelitis were found. Leichenstern has noted the existence of periosteitis and osteomyelitis of the tibia and fibula following influenza. He quotes Bose as mentioning a case of purulent osteitis of the tibia in a young man. In another reference Franke mentions a case of osteomyelitis of the sternum following influenza, in which syphilis could probably, but not absolutely, be ruled out. In the Surgeon General's Office, there is made mention of a case of osteomyelitis of the temporal bone as a sequel to influenza. Nichols, in his exhaustive study, makes no mention of osteomyelitis following influenza

The history of five cases follows:

Case 1. B. F., age 9. During the height of the epidemic of influenza, I was called in consultation to see the patient. At that time he had incisions on the forearm which had been made by V. A. Loeb. The history was that following an attack of influenza and pneumonia during which the child was very sick, a swelling of the forearm appeared and spread rapidly. There seemed to be solution of the superficial fat. I advised then that the incisions on the arm be made larger to allow better drainage. About two weeks afterward, I was again called by Dr. Loeb to see the patient who then had a metastatic abscess around the left scapula. The child was removed to the Jewish Hospital where the abscess was opened and the sinuses of the arm were enlarged and curetted. After discharge from the hospital I did not see him until 9 weeks later when examination revealed an enormously swollen arm, with atrophy of the muscles of the arm and shoulder girdle. The forearm resembled a leg and there was practically no motion at the elbow-joint or the wrist-joint. The forearm could not be used because of the swelling and the excessive weight. Small quantities of pus were discharged from the sinuses. Diagnosis: acute osteomyelitis of the bones of the forearm.

A roentgenogram was taken and revealed an extensive osteomyelitis of the entire radius with a small focus of necrosis of the olecranon and the condyles of the humerus (Figs. 2 and 3).

Removal of the radius was then advised but before operating we consulted J. C. DaCosta, who agreed that all the dead bone should be removed. This was accordingly done. An incision was made the entire length of the forearm and on reaching the radius we found that the bone was lying in its

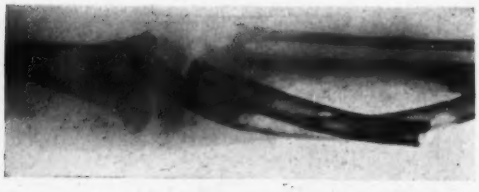


Fig. 1. Case 5. Note that the roof of the tibia has been removed.

bed practically as a huge sequestrum. It was easily removed. A small portion of the articulating surface of the radius including the lower epiphysis was left so as to prevent encroachment on the wrist-joint (Fig. 4).

At the present time the patient has an incurvation of the hand due to the removal of the radius; he has some supination and pronation and the elbow can be bent to an angle of 45 degrees. It was proposed at a later date to graft a piece of bone to prevent the inevitable deformity that must result from the removal of the radius, but this may not be necessary now on account of the regeneration of the radius which has since taken place (Fig. 5).

CASE 2. Six weeks before the patient was seen in consultation with Dr. Applebach, he had suffered an attack of influenza. The mother states that one evening after supper the child suddenly complained about pain in the knee-joint and inability to walk. When I examined the child there was considerable swelling above the left knee-joint. A diagnosis of osteomyelitis of the femur was made. The patient was taken to the Jewish Hospital where an X-ray examination confirmed the diagnosis. An incision was made on both sides of the thigh above the condyles of the femur where pus was obtained. Through-and-through drainage was used. The child made an uneventful recovery, perfect healing and recovery taking place in 8 weeks (Fig. 6).

CASE 3. A woman about 30 years old, with symptoms of influenza, was admitted to the Mt. Sinai Hospital on Dr. Shmookler's service. While in the hospital she developed swellings of both forearms and the right elbow-joint. These swellings were similar to those in Case 1, but the further progress of the case demonstrated that there was a periosteitis instead of a deep infection of bone. The swellings were incised and a large amount of pus was liberated. The wounds healed well.

The last two cases were fatal.

CASE 4. H. L.; age 16. Several weeks before admission symptoms of influenza developed. The patient was extremely emaciated. He had symptoms indicating osteomyelitis of the lower end of the femur and this diagnosis was verified by X-ray examination (Fig. 7). At operation very little pus was found but the general toxæmia was so severe as to offset any benefit derived from the incision and drainage. An attempt was made to increase his

Fig. 2. (at top) Case 1. Necrosis of radius, bismuth injection. Anterior view.
Fig. 3. Case 1. Same as Figure 2, lateral view.

resistance by forced feeding and blood transfusion but to no avail.

CASE 5. A baby one year old was admitted to the Mt. Sinai Hospital with symptoms of osteomyelitis of the tibia following influenza. This little patient was first operated upon in another hospital. An abscess was opened over the shaft of the tibia. X-ray examination disclosed a deep infection of the bone. The entire roof of the tibia was removed. The medullary cavity was exposed and from it exuded a large amount of pus. A few days afterward a large swelling appeared above the knee-joint and the head of the tibia. This was opened and a communication was established between the knee-joint and the head of the tibia. The child developed diphtheria. She was removed to the Municipal Hospital and died suddenly the evening of admission.

Excision of the radius may be considered a rare operation although in a search of the literature 20 cases have been recorded. Smith reports a case in a child of 8. The patient had had measles 4 years before the onset of the present trouble. On account of the swelling of the arm the child attended hospitals for two years being treated for sinuses. These seemed to be tuberculous in character. The carpal epiphysis was not removed. The patient used his arm freely and was able to write.

However, the honor of first having removed the radius is claimed by Carnochan, and he claims to have been the first also to excise the ulna. This was in 1855. The patient, a male, aged 18, had received treatment for months without benefit when excision of the radius was performed. Following the opera-

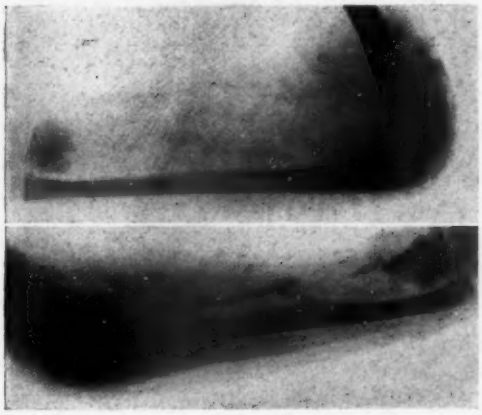


Fig. 4. (at top) Case 1. The radius removed.
Fig. 5. Case 1. Beginning regeneration of the radius.

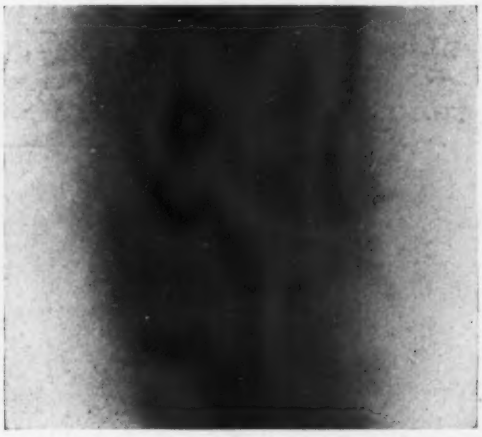


Fig. 6. Case 2. Necrosis of lower end of the left femur.

tion the patient had good motion, was able to write with a pen, and had good muscular power. He followed again his occupation of laborer. No cause has been assigned for the necrosis of the radius.

According to the literature Smith, in 1848, in the case referred to above, was the first to remove the radius for necrosis.

Fuqua reports the case of a negro child 10 years old with necrosis of the entire shaft of the radius as a result of a fracture two years before. The radius was removed, the entire periosteum remaining. A rudimentary radius was reproduced. The child had good pronation and supination, flexion and extension. Fuqua quotes Gross as saying that, to his knowledge, up to 1859 the entire radius had not been removed. In 1863 Gross removed the entire radius subperiosteally for necrosis following a gunshot wound. The patient recovered and had a useful hand and arm.

Wright says that one rarely finds necrosis of the entire length of long bones. He reports the case of a boy 12 years old who had a fall two years before consultation. Part of the radius had been removed and a year later the remainder of the radius was removed. The patient was able to scratch his head, lift a chair, carry a weight of 15 pounds, feed himself, open a door and write his name.

In 1881 Field referred to Carnochan's case as the only one on record. Field's patient was a boy who after an injury developed fistulous openings. After operation the strength in his arm was good. His infirmity did not interfere with his work and he was able to carry large buckets of water.

Butcher reports the case of a boy of 17, who had suffered a fracture of the lower epiphysis. After he had been treated about a year with various topical applications and constitutional remedies, the radius was removed. The patient could then flex the fingers, and pronation and supination were well done.

Chavasse has had a rather unique experience. His patient suffered from necrosis of the radius from infancy. Sequestrotomy was performed nine times. When the patient was 18 the radius was removed. He was able to feed and wash himself and carry an ordinary bucket.

Belcher reports the case of a boy 9 years old who was admitted to the hospital for sinuses following necrosis of the radius. The radius was removed in two sections. There was no regeneration of the radius.

Carnochan reports the case of a male, age 20, who, following an accident, was treated for three months. The elbow-joint was immovable. In 11 weeks he was discharged

Fig. 7. Case 4. Necrosis of lower end of femur.

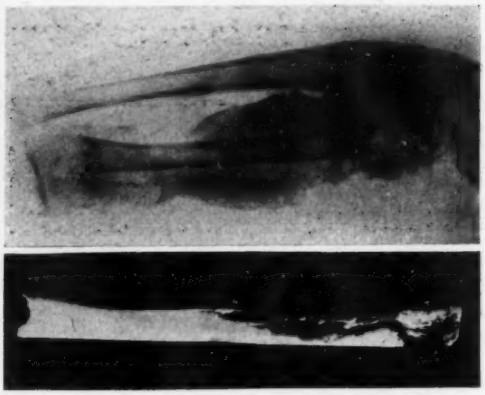


Fig. 8 (at top). Necrosis of the radius. P. G. Skillern's case.
Fig. 9. Sequestrum of radius. P. G. Skillern's case.

cured, and was able to perform the duties of an orderly. Radial disfiguration was present.

Cutter reports the case of a scrofulous child who was compelled to have the radius excised; some of the fingers had been removed at a previous operation.

Chapin reports the case of a negro boy, age 14, who suffered from tubercular sinuses of the arm. The radius was excised at the epiphysis of the wrist. Chapin refers to Velpeau (1839) who always advised amputation in such cases. Quite a controversy arose as to which was the better operation, amputation or resection of the radius. The consensus finally was that excision was the operation of choice and the various reports as to the ultimate function of the arm certainly bear this out.

Duffield cites a case of spontaneous tuberculosis of the radius which was later excised.

Galozzi reports a patient who had been treated for eighteen months without result. The entire radius except the lower portion became necrotic. The periosteum regenerated new bone around the sequestrum. The latter was removed. The wound healed and the patient had a useful arm.

Carl Beck, in 1893, reported a case of regeneration of the radius after its removal for osteosarcoma.

A. C. Godfrey performed excision of the radius for tuberculous disease. For ulnar dislocation from the carpal bones, he recommends a steel support with a hinge corresponding to wrist movements.

P. G. Skillern, Jr., reported the case of a child of 3, who previously had an osteomyelitis of the femur which healed. Necrosis of the radius followed. There was complete regeneration of the radius after its removal. I am indebted to Skillern for the privilege of using his illustrations. These represent exactly the condition as found by the various surgeons whose cases are cited in this paper (Figs. 8 and 9).

Tavernier excised the radius after a low amputation of the arm, following an explosion of dynamite. The excision was performed about two months after the amputation on account of necrosis of the radius. While pronation and supination were diminished, flexion and extension of the elbow were entirely conserved.

The encouraging reports as to the return of function in these cases is quite remarkable. The case reported by the writer shows a return of function also but not to the same degree as that shown by other writers. While the child is able to write and hold his knife and fork, still the normal functions are greatly reduced partly on account of atrophy of the muscles of his hands and arm and a

Fig. 10.

Fig. 10. Case 1. Typical radial deformity following excision of radius.

Fig. 11.

Fig. 11. Case 1. Apparatus designed to overcome the radial deformity.

partial ankylosis of the elbow-joint due to necrosis also of the olecranon process and the condyles of the humerus. There is also present the typical radial deformity noted by various writers. To counteract this deformity the patient wears a steel support as recommended by Godfrey (Fig. 11).

The table of cases in which excision of the radius was performed follows:

Surgeon	Cause	Age of patient	Date of operation	Function
H. Smith	Tuberculosis	12	1848	No report
Carnochan (S. Smith)	No report	18	1855	Good
Belcher	No report	9	1857	No report
Cutter	Tuberculosis	No report	1857	No report
Butcher	Accident[1]	17	1859	Good
Carnochan	Accident	20	1859	Good
Chappin	Tuberculosis	14	1861	Good
Gross	Accident	No report	1863	Good
Galozzi	No report	19	1867	Good
Fuqua	Accident	10	1878	Good
Field	Accident	No report	1881	Good
Duffield	Tuberculosis	10	1880	No report
Wright	Accident	12	1885	Good
Chavasse	No report	19	1885	Good
Beck, C.	Sarcoma	18	1893	Good
Godfrey	Tuberculosis	24	1893	Good
Tavernier	Accident	No report	1913	Good
Skillern	Septicæmia	3	1918	Good
Behrend	Epidem. influenza	9	1919	Good
Erichson[2]			1855	

[1]The accident was followed by necrosis of the radius for which the excision was performed.

[2]No description of this case could be found in the library of the College of Physicians.

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THE FREQUENCY AND SIGNIFICANCE OF OMPHALITIS

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At one time all inflammatory lesions of the umbilical cord were attributed to syphilis. Subsequently this view was discredited, for in many instances omphalitis was demonstrable in cases in which the mother gave no history of syphilis and the familiar anatomical lesions were not present in the fœtus. It has been shown also that in the presence of congenital syphilis, the umbilical cord is very rarely infected with the treponema pallidum. On the other hand, thrombosis of the umbilical vein has been recorded in a case of intrapartum infection in which streptococci were found in the placenta and in the cord. Thus suspicion was directed toward bacterial infection as an important cause of omphalitis, and the problem has been attacked from this angle in the present study. The frequency of omphalitis, the nature of the lesion, its relation to placental bacteriæmia and to fœtal mortality, are all questions of interest especially in view of the great effort now directed toward reducing fœtal mortality.

On microscopic examination of the cord in 2,200 consecutive cases in which the infant weighed more than 1,800 grams, 43 showed leucocytic infiltration of the vessel walls and the adjacent connective tissue. To trace the source and causative factor of this lesion additional sections, both toward the fœtus and toward the placenta, were studied. The clinical results for the mother and for the infant have been tabulated, the histories being analyzed to learn what significant features these cases present.

The lesion consists of an extravasation of polymorphonuclear leucocytes into the wall of the umbilical vein, occasionally into the wall of one or both of the arteries and into the whartonian jelly. Outside the vessels the leucocytes are diffusely distributed in the interstices between the connective-tissue fibers, more compactly at the center of the cord and less densely at its periphery. The lesion varies in severity. It may pertain only to a segment of the vein wall or, at the other extreme, may involve all three umbilical vessels. Rarely thrombophlebitis exists. The lining of the affected blood vessel presents

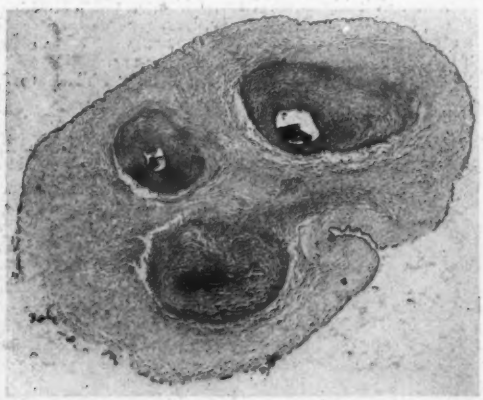

Fig. 1. Normal cord. x6.

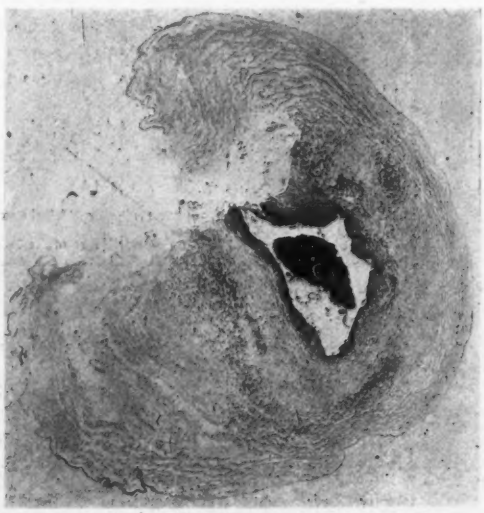

Fig. 2. Portion of affected cord, showing thrombus in vein and adjacent whartonian jelly infiltrated with polymorphonuclear leucocytes.

no increase in thickness, although at times there is dissociation of the connective-tissue fibers of the intima. In two cases perivascular necrosis was present. Typically, the inflammatory exudate consists of polymorphonuclear leucocytes, loosely disseminated, but in two instances these cells were so densely massed as to resemble early abscess formation. On the other hand, occasionally, mononuclear elements were conspicuous, at times proportionately equal to the polymorphonuclear cells.

Frequently a phagocytic macrocyte is present. This large cell with considerable protoplasm and a spherical, deep-staining, granular nucleus appears at the periphery of the cord, wandering in the loose connective tissue beneath the epithelium. The presence of these cells is not peculiar to the lesion; they are also not uncommon in the loose subamniotic connective tissue of the placenta. Their protoplasm is granular and varies in shape from diamond to rhomboid. The rôle these cells play, not clear at present, is nevertheless a conspicuous one and merits further investigation.

Omphalitis, as has been said, was once ascribed to syphilis. In 1903 Bondi reported a study of 35 cords of which 15 occurred in syphilitic patients and showed the characteristic white cell infiltration in the vessel walls of the cord. The other specimens, obtained from patients with non-specific infectious or constitutional diseases, were normal. Therefore, he ascribed this cord lesion to syphilis and stated that the accepted signs of the disease, such as endarteritis and proliferation of the stroma of the cord were rarely present. This view was adopted later by

Fig. 3. Infiltration in vein wall and surrounding connective tissue diminishing toward the periphery of cord.

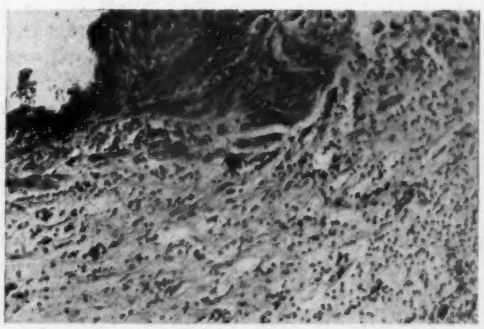

Fig. 4. Polymorphonuclear leucocytes infiltrating vein wall.

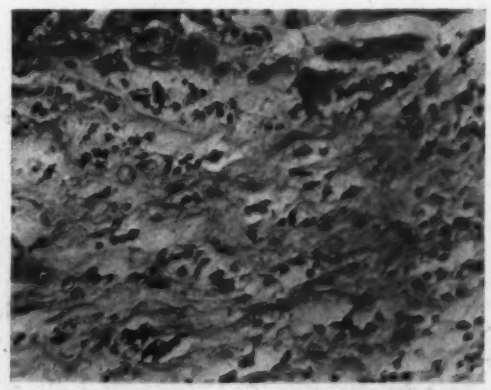

Fig. 5. Dissemination of leucocytes in interstices of connective tissue. High power.

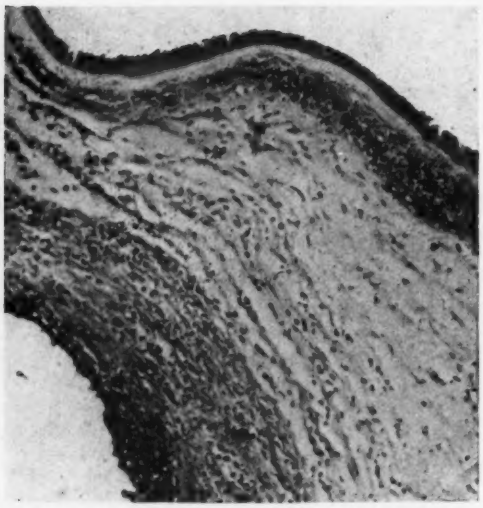

Fig. 6. Fœtal surface of the placenta, with amniotic epithelium, branch of umbilical vein and leucocytic invasion of subamniotic connective tissue.

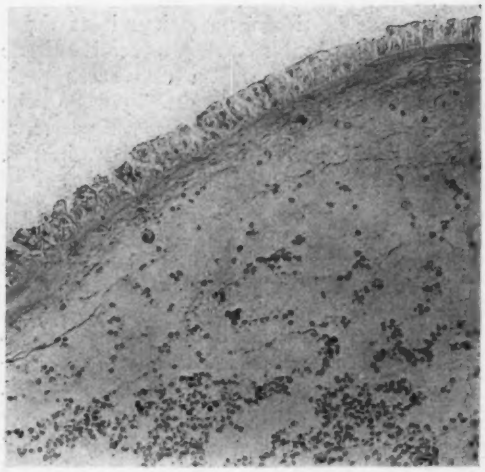

Fig. 7. Amniotic epithelium, showing distortion after rupture of membranes. "Macrophages" and leucocytes in the loose subamniotic connective tissue.

Thomson who reported a series of 59 cases, one of which showed the characteristic lesion in the cord although the patient denied luetic infection. Thomson, however, regarded the clinical history as less reliable than the microscopic findings Subsequently Thomson and Boas made a control study of 500 cases, the result of which, they considered, served to emphasize Bondi's original statement.

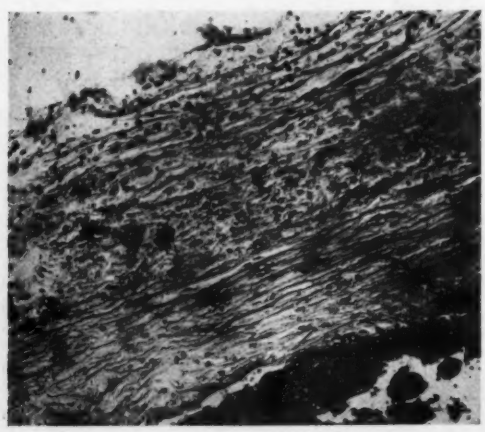

Fig. 8. Sub-amniochorionic connective tissue zone with loss of the epithelium and invasion by leucocytes.

Von Winkel's *Handbook of Obstetrics* and Ziegler's *Pathology* make conservative statements to the effect that the lesion is chiefly syphilitic and point the need for further investigation. In 1912 Simmonds reviewed the subject and denied the specificity of the lesion for syphilis, but suggested no other causative factor. However, he was impressed by the fact that a great number of these patients ran a febrile course in the puerperium.

Among our 43 cases of omphalitis, there were only three mothers who presented a positive Wassermann test as well as a typical histological picture of placental syphilis. On the other hand in the entire series of cases (2200) which are the basis of this report, there were 29 instances of maternal syphilis without any lesion of the umbilical cord. Furthermore in 40 instances of omphalitis, there was no evidence of syphilis.

There were 3 cases of omphalitis in which the mother was syphilitic. The first of these was delivered spontaneously of a child which died on the second day from hæmorrhage of the newborn. The mother had a febrile puerperium. The second entered the hospital after rupture of the membranes, with a temperature of 100° F. and was delivered of a stillborn fœtus. The third entered the

hospital late in the second stage, with a temperature of 99.6° F. and was delivered of a living child. Her temperature rose to 101° F. on the third day on account of a mild puerperal infection.

The statistical evidence just presented demonstrates that syphilis is not commonly attended with inflammation of the umbilical cord. Furthermore, in the majority of cases in which the cord lesion occurs, the mother is not syphilitic. In view of these facts we believe that, in cases presenting syphilis and inflammation of the umbilical cord, two diseases rather than one must be reckoned with. Omphalitis, it is clear, appears so frequently in cases without any sign of syphilis, that its cause must be sought elsewhere. Fortunately, its origin need no longer be left to conjecture. It depends, we find, upon bacterial invasion of the placenta, and the latter lesion generally occurs in cases in which the membranes have ruptured prematurely.

Infection of the contents of the pregnant uterus, as Hellendall has shown by animal experimentation, may occur by three routes: (1) the transamniotic, usually with, but occasionally without, the previous rupture of the membranes; (2) by way of the maternal blood as in smallpox; and (3) by way of the fallopian tube from the abdominal cavity, as in appendicitis. To this statement Warnekros added that in cases of transamniotic infection of the placenta, two mechanisms are possible: (a) that by which organisms are transmitted through the amniotic fluid directly to the fœtal surface; and (b) that by which they proceed from the point of rupture of the membranes along their surface to the nearest placental margin. Slemons has reported cases of placental bacteriæmia in which organisms were found invading the subamniotic connective tissue. These cases showed also the typical leucocytic invasion of the cord; in one there was thrombosis of the umbilical vein.

Since the lesion of the umbilical cord, as originally described by Bondi, accompanied the subamniotic invasion reported by Slemons the next step was to discover what relation, if any, the two might have. Sections through the insertion of the cord upon the placenta were made. At this point the fœtal vessels divide to spread over the fœtal surface of the organ. In every case of omphalitis, the branches of the umbilical vein at the root of the cord were involved in an inflammatory reaction, but the arteries of this area were involved in only one instance. The course of the inflammatory process could be traced backward along the veins into the subamniotic connective tissue. The direct continuity of the lesion, just indicated, was demonstrable in 38 of the 43 cases of omphalitis. In the 5 negative cases, a limited quantity of placental tissue had been preserved and its study was correspondingly restricted. The evidence at hand, therefore, indicates that generally, if not always, omphalitis is preceded by bacterial infection of the placenta.

The latter lesion, as I have said, is prone to occur in cases where the membranes rupture prematurely. Here, the retraction of the uterus preceding delivery reduces the size of the amniotic cavity, forces the amniotic epithelium to change from cuboidal to a high columnar type and restricts the basal attachment of the cells. The nuclei pressed upward toward the surface of the amnion are occasionally expelled from the cells which consequently undergo necrosis. Obviously, injuries of this kind impair the protective action of the epithelium against bacterial invasion and, if bacteria are present in the amniotic fluid, they may enter the subamniotic connective tissue and the walls of the fœtal vessels which cross the placenta. Premature rupture of the membranes was noted in 26 cases, or 60 per cent of the cord infections. In 13 of these cases, rupture of the membranes occurred from 18 hours to 5 days before the onset of pain.

It is well known that any factor which tends to lengthen the duration of labor increases the risk of infection. From this viewpoint, the clinical histories of the cases of cord infection present certain significant features, for not infrequently the familiar causes of the prolongation of labor were present. Thus, 16 were primiparæ, and 13 had contracted pelves causing dystocia, though only 2 of these required operative assistance. In the series there were three breech pre-

sentations, three persistent occiput posteriors and one transverse presentation. In 25 cases the delivery was spontaneous and in 18 an appropriate obstetrical operation was performed. The duration of labor was prolonged in 25 cases, including 13 of the primiparæ, which represents 58 per cent of the cases in which omphalitis occurred.

Some degree of fever during labor, an outstanding feature of infection, was noted in 22 cases of omphalitis. The temperature varied from 99 to 102° F. In some instances the temperature was not recorded and in others an observation was made only at the very onset of labor. While these data are incomplete, our experience teaches that, in the presence of intrapartum fever, infection of the placenta is the rule and frequently the umbilical cord becomes involved. If fever occurs in conjunction with premature rupture of the membranes and prolongation of labor, infection of the placenta with extension to the cord becomes almost certain.

The ultimate result for the mother and for the infant in cases of intrapartum fever becomes a matter of the first importance. For the mother it appears that the danger is greatly reduced by the expulsion of the placenta, and usually the temperature falls when the delivery is complete. On the other hand maternal infection may declare itself 48 to 72 hours after delivery. This was noted in 11 of the cases of omphalitis, but the puerperal sepsis was mild, with no maternal deaths.

For the infant the results are more serious. Fourteen infant deaths occurred among the 43 cases of omphalitis, yielding a mortality of 32.5 per cent. There were 8 stillbirths; 3 infants died on the first day after birth, 2 on the second and 1 on the fourth day. This very high mortality, it must be made clear, cannot be attributed to the cord lesion which of itself may become the cause of death only when the cord vessels are thrombosed. Generally, we believe, the death of the fœtus or of the infant is attributable to infection of the fœtal blood, although at times it is impossible to determine what rôle an operation to effect delivery has played in the death of the infant. One fœtal death followed a placenta prævia; in 3 additional cases, lacking autopsy findings, the question of the cause of death remains undifferentiated between infection and injury. The remaining fatalities we ascribe to placental bacteriæmia. If 23 per cent of these intrapartum infections were fatal to the fœtus, it is of considerable moment, and precaution must be taken to reduce the frequency.

TABLE OF INCIDENCE AND MORTALITY OF INFECTION OF THE UMBILICAL CORD

Group	Number of Cases	Cord Lesions	Incidence Percentage	Deaths	Mortality Percentage
A	1200	26	2.1	11	42.3
B	1000	17	1.5	3	17.6
Total	2200	43	1.9	14	32.5

The value of precaution in the conduct of labor is proved by these statistics which may be classified chronologically into two groups: (a) the earlier, including 1,200 cases, pertains to a period when placental bacteriæmia was given no special consideration; (b) the later, consisting of 1,000 cases where strict precaution was observed—especially the limitation of vaginal examinations—in prolonged labors and after the rupture of membranes except as a preliminary to some operative procedure to effect delivery. In the first group 26 cord infections were encountered with an infant mortality of 42.3 per cent while in the second group there were 17 cord infections and a mortality of 17.6 per cent. This decrease in the number of infections as well as in their severity we ascribe chiefly to the substitution of rectal for vaginal examinations.

SUMMARY

In conclusion the results of this study may be summarized as follows:

1. In a series of 2,200 consecutive deliveries, an inflammatory lesion of the umbilical cord has been found in 43 cases.

2. The lesion is not pathognomonic of syphilis, for (a) it was present in 40 cases where there was no evidence of syphilis, and (b) it was absent in 29 cases of undoubted syphilis.

3. The lesion arises by the extension of bacterial infection from the placenta.

4. Bacteria are frequently demonstrable in sections of the cord.

5. The lesion is commonly associated with prolonged labor after premature rupture of the membranes.

6. The frequency of these infections and the resulting infant mortality may be reduced by the use of rectal in place of vaginal examinations.

NOTE:—The following pertinent case has been treated in the clinic since this paper was completed.

After a prolonged labor a primipara was delivered spontaneously of a living child. Its temperature frequently reached 101° and it took nourishment poorly. On the eighth day blood cultures were positive for streptococci. Death occurred on the eleventh day and at autopsy empyema and purulent pericarditis were found. Streptococci and bacillus coli were isolated. Sections of the placenta and cord showed the lesions described associated with streptococci. The mother had a mild grade of fever on the third and fourth days postpartum but otherwise had an uneventful convalescence.

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RUPTURE OF THE RECTUM DURING LABOR

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THERE are numerous instances in the literature of rupture of the rectum but in a careful search of the standard books on obstetrics and rectal diseases, I have been unable to find a case occurring during labor. In a personal letter from Joseph B. De Lee, of Chicago, he stated that he has never seen a similar case.

Wallis (1) states that certain rectal strictures are due to pressure of the child's head during labor; Fulton (2) reports a positive case of stricture of the rectum following a prolonged labor; Tuttle (3) mentions perforation of the rectum in cases of stricture of the rectum due to syphilis, but no mention is made as to its occurrence during delivery.

As syphilitic stricture of the rectum occurs mostly in women, it is remarkable that this accident is not more frequent during childbirth. It can be readily seen that as the rectal wall above the stricture is dilated and weakened, and that as there is generally a condition of coprostasis present, the pressure of the fœtal head descending would cause a pressure on the accumulated fæcal material which, unable to pass downward, because of the stricture, bursts through the rectal wall.

In the case herein reported we were in the dark as to the exact condition. Rupture of the uterus was suspected. As the woman was in such a profound state of shock, an exploratory laparotomy was contra-indicated. A rectal examination threw no light on the condition as the perforation was far beyond the reach of the examining finger.

Patient, female, age 37, entered hospital in a state of profound shock. Pulse 156, temperature 102, respiration 42. Skin gray, lips cyanosed, and face covered with perspiration. Abdomen greatly distended. She gave the following history:

Family history: The mother and father are living and well. The husband is in good health. The patient has had 3 children, all living and well.

Present trouble. The patient was delivered at her home 30 hours prior to entrance to hospital. A low forceps operation was performed and almost immediately after the delivery of the child the patient complained of severe pain in her left side and soon afterward began to show signs of shock. Twelve hours later she developed abdominal symptoms and was sent to the hospital. The general physical examination was negative. The abdomen, as stated before, was greatly distended, tympanitic except over uterus which gave a dull area 1 hand's breadth above symphysis. The distention of the abdomen was so great that the skin was broken in several areas. The vaginal examination revealed a soft, boggy, puerperal uterus, and a recent bilateral laceration of the cervix, also a recent perineal laceration. Slight lochia but no odor, and negative on microscopic examination. Leucocyte count 21,000. Rectal (low) examination was negative.

Subsequent history. No definite diagnosis was made except that there was a peritonitis present. Three hours after entrance the patient had an almost imperceptible pulse. Temperature 104.8°, body surface cold and clammy. Condition of patient gradually became worse and she died 12 hours after entering hospital. Her last temperature before death was 107°.

Postmortem examination by Dr. F. A. Baldwin. Acute diffuse fibrinous peritonitis; rupture of rectum at the rectosigmoidal junction; old stricture of rectum (syphilitic); postpartum uterus; cloudy swelling of liver and kidneys; acute congestion of spleen, œdema of lungs; acute dilatation of heart; no perforation of vagina; appendix negative; recent laceration of perineum.

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INFECTIONS OF THE KIDNEY IN GYNECOLOGIC PRACTICE[1]

By W. C. DANFORTH, B.S., M.D., F.A.C.S., Evanston, Illinois

THIS paper is presented not with the idea of contributing anything original but solely with the purpose of bespeaking a more accurate clinical observation of possible lesions of the urinary tract than these sometimes receive. During the past 15 months there have come under the observation of the writer 25 cases of pyelitis of which one was bilateral, 3 cases of pyonephrosis, 1 case of congenital cystic kidney, 1 case of obstruction of the ureter by an aberrant renal artery, 2 of tubercular kidney, and 1 case of pyelitis associated with imperforate vagina and absent uterus. Of these cases of pyelitis all were of colon bacillus origin but 2, 1 of which showed a growth of pneumococcus and 1 a growth of staphylococcus. This latter can very well have been a chance contamination as staphylococcus may be found as inhabitants of the normal bladder.

It seems therefore that the gynecologist must find himself at times trespassing upon the field of the urologist. Kidney infections in women are so common that anyone who sees very many gynecological cases cannot fail frequently to see women in whom infections or other lesions of the kidney are present. It is estimated that pyelitis occurs four or five times as frequently in women as it does in men and when we take into consideration the frequency of this lesion during pregnancy the ratio does not seem excessive.

In considering etiology there does not seem to have been sufficient stress laid upon infections of the urinary tract which occur in infancy. About two years ago, following a suggestion of Helmholz, who had within a short space of time seen several very young babies with cystopyelitis, it was made a routine in the gynecologic-obstetric service of the Evanston Hospital, to obtain samples of urine from all infants in the ward service as soon as possible after birth and from all infants of my own private patients. Since that time the number of instances in which pus has been found in urine of infants of a few days old has been striking. It seems fair to assume that many unrecognized and there-

[1] Read before the Chicago Gynecological Society, November 21, 1919 (For discussion, see p. 316.)

fore untreated infections of the kidney in infancy may be the starting point for urinary lesions of a more serious character in later life, and it is suggested that a careful routine observation of urinary findings during the first week of life in maternity hospitals and the active routine treatment of urinary infections revealed thereby would serve as a preventive measure of considerable value.

As to the route through which infections may reach the kidney, we may consider the following:

1. Ascending infections through the lumen of the ureter. The possibility of infections arriving in the kidney by this means must be admitted. Cystograms have shown that fluid in the bladder may travel upward into the ureter, particularly if the latter be dilated. That a retrograde peristalsis may occur in the ureter comparable to that occurring in the intestine in cases of intestinal obstruction, is evidenced by an observation of Kretschmer that a stone which has traveled down to the lower levels of the ureter may again be propelled upward and found again on later radiography to lie at a considerably higher level than that at which it had previously been observed. While this is probably not a frequent mode of transmission of infection it must be regarded as a possibility.

2. Transmission upward of infection through the lymphatics and the wall of the ureter. This possibility was pointed out some years ago by Bauereisen and the same mode of transmission of infection was experimentally studied some years later by Eisendrath. Cabot and Crabtree, however, two years ago, suggested in arguing against this mode of transmission, the anatomical fact that in regions of the body where the course of the lymphatics has been demonstrated, it has been found that they in general have followed courses pursued by the blood vessels supplying the region. While the actual course of the ureteral lymphatics has, I believe, not yet been demonstrated, it is known that the blood supply of the ureter is a segmental one, that is, its vessels traverse its wall for a certain distance and then leave it. If the lymphatics follow their usual rule in following the blood vessels of the ureter, they would therefore be prone to leave the wall of the ureter before arriving at the level of the kidney. Infection, therefore, could scarcely travel in an unbroken path the length of the ureter through the lymphatics.

3. The lymphatic connection between the cæcum and ascending bowel has been pointed out by von Franque, a route by which infection may pass directly from the bowel to the ureter. A certain relationship between apendicitis and lesions of the right kidney has been suggested by some of the French writers.

4. By all means the most common route by which infection reaches the kidney is the blood stream. As colon bacilli are always present in enormous quantities in the large bowel, they must be present at times, if not constantly, in the blood stream. Bacteria are excreted by the kidney parenchyma without damage to the latter under normal circumstances. When, however, the drainage from the kidney is impeded or the individual's resistance may be lowered, infection may occur.

Any one who has had occasion to observe the urine in large numbers of cases of pregnancy is aware of the frequency with which pyelitis occurs in these cases, the exciting factor being pressure upon the ureter by the pregnant uterus. It may also, I think, be safely stated that many women pass through pregnancy with an infected kidney of mild degree which fails of recognition. A very large percentage of these cases, because of cessation of treatment after the pyelitis has been symptomatically relieved, which usually occurs after labor, or because it has not been recognized at all, are not followed up to the point of bacteriological cure, hence many of them may later develop an attack of pyelitis. Cabot and Crabtree have pointed out that non-tubercular infections of the kidney are divided into three classes: those of bacillary origin, caused by bacilli of the colon group, which are prone to limit themselves, at least at first, to the kidney pelvis. These are the most easily curable and by all means the most frequently seen in women. Second, those of coccal origin which select the kidney parenchyma, and third, those of mixed coccal and bacillary character which may attack both pelvis and parenchyma.

These infections the gynecologic surgeon is constantly seeing. Frequently a confusion as to diagnosis arises between pyelitis and appendicitis, many cases of kidney infection being looked upon as inflammation of the appendix. It has happened recently in my own experience to be asked twice in one week by two different physicians to see women in whom an acute appendicitis was assumed to be present, in both of whom the real lesion was a colon bacillus pyelitis which was easily demonstrated by urinary examination and ureteral catheterization and both of which were promptly relieved by appropriate treatment.

A certain resemblance in the symptomatology which may occur in these two processes may easily deceive the unwary. The usual symptoms of pyelitis are pain in the infected kidney together with tenderness in the corresponding costovertebral angle, frequently fever, elevation of leucocyte count, pain extending along the infected ureter with tenderness over the ureter in the lower abdomen. When the lesion is on the right side, particularly if nausea accompanies it, which is sometimes the case, the hasty observer may easily conclude that it is the appendix which is involved. Right costovertebral angle tenderness and the presence of pus in a catheterized sample of urine will suggest the possibility of kidney infection which may easily and quickly be proved by the use of the cystoscope. No woman should be operated upon for acute appendicitis unless the appendicitis be a very clearly defined attack, until the possibility of an acute pyelitis is eliminated.

During pregnancy it has long been a rule that no appendectomy should ever be done without a previous microscopic examination of the urine. The abdominal surgeon may also well afford to follow a similar precaution.

As to the treatment of pyelitis, my own experience has brought me to the consideration of but one means of treatment; namely, lavage of the kidney. It seems fair to say that in cases of simple pyelitis in which the parenchyma of the kidney is not involved, a cure may practically always be attained by this means. Much argument has been had as to whether it is the simple drainage by means of the catheter and the possible widening of the lumen of the infected ureter, the mucosa of which may have been swollen as a result of infection, by the mechanical pressure of the catheter, or whether it is the use of an antiseptic solution which is deposited directly in the infected area. My own opinion is that the antiseptic plays an important part. In mild pyelitis, however, associated with a severe cystitis, lavage should not be practiced until the cystitis has been treated and its severity lessened, if possible.

Treatment of the pyelitis should also be associated with relief of constipation, establishment of regular bowel drainage, and the administration of bulgarian bacillus to reduce the activity of the colon bacillus in the bowel as far as possible.

The kidney is irrigated at intervals of four days with a solution of nitrate of silver varying from 1:300 to 1 per cent, the number of lavages necessary varying from 1 to 6, but are oftenest 3 or 4. The very frequent practice of treating what appears to be a cystitis by medication by mouth or vesical irrigation without any definite attempt to discover from whence the cellular elements in the urine come, cannot be too strongly condemned.

It is possible that the very recent work of Young, White, and Swartz of the Brady Institute at Johns Hopkins with mercurochrome-220, may be the means of putting us in possession of a new and valuable drug for use in infections of the urinary tract.

The criterion of the cure is not the appearance of clear urine or one in which no cellular elements are to be observed microscopically. Certain evidence of cure is only at hand when at least one, or better two, successive urinary cultures fail to show the presence of bacteria.

An illustration of the consequence of failure to recognize infections of the kidney may be illustrated by the following case:

A woman of 42 on the occasion of her last labor, nine years previously, states that she had a cystitis. She does not remember having had pain in the back during pregnancy. The cystitis was treated by vesical irrigation on several occasions and in the past few years she has had further attacks of bladder infection which have been treated in a similar way. During the past year she has constantly had trouble with the bladder with some discomfort in

the right lumbar region and has been losing weight. Recently she came under the observation of another physician, who, for the first time, recognized that there was a mass palpable in the right upper abdomen. Upon my seeing her I agreed that this was undoubtedly a pyonephrosis, as the urine was heavily loaded with pus, and upon cystoscopy this was easily demonstrated, the infective organism being the colon bacillus. A functional test showed the other kidney to be in excellent condition. Nephrectomy was carried out removing a large pus kidney containing five stones. This woman undoubtedly had had during pregnancy an infected kidney, which, had it been taken care of at that time and treatment continued during the puerperium to the complete recovery, might perhaps have saved her kidney.

An illustration of the ease with which obscure lesions of the kidney may be overlooked and of the truth of the rule that neurosis should only be assumed when painstaking study fails to reveal a real cause for symptoms, is illustrated by the following:

A girl of 19 entered the medical service of the hospital complaining of pain in the right side of the back. She gave a history of having recently been in one of the large hospitals of the city where she had remained for about two weeks and had been discharged after having been told that she was a neurotic. While the girl was unquestionably nervous, the medical man under whose observation she was, was quite convinced that neurosis did not explain the pain, inasmuch as it was always constant in its location and suggestion would not cause her to complain of pain elsewhere. Tenderness was invariably present over the right costovertebral angle. Examinations carried out on other parts of the body, such as the apices of the lungs, with the idea of distracting her attention, followed by sudden percussion over the painful area never failed to elicit complaint. The nurses while making her bed and otherwise caring for her were instructed to strike her with apparent inadvertence in various parts of her anatomy, but without result except when the right costovertebral angle was attacked when she invariably complained of pain. The urine was absolutely negative, cystoscopies were negative and catheters passed into both kidneys without trouble. Urinary cultures from both kidneys were negative and it was only when a pyelogram was made showing that the kidney would hold between 13 and 14 cubic centimeters of fluid, that the nature of the lesion began to be suspected. She had a hydronephrosis, which, without any history of infection or obstruction in the ureter, seemed difficult to explain. A diagnosis was, however, ventured of an obstruction of the ureter due to an aberrant renal artery, in spite of the fact that the pyelogram showed no kink in the ureter. She was then operated upon by a colleague on the surgical service, exposure of the kidney showing an aberrant artery about the size of a goose quill entering the kidney at the lower pole over which the ureter was looped. The kink in the ureter was undoubtedly straightened out by the catheter and so did not show in the plate. After operation the pain disappeared and a report received recently, four months after operation, indicates that she is entirely free of pain.

One other interesting case was seen, that of a girl of 22, who was referred for pyelitis.

On examination she was found to have an imperforate vagina, there being merely a pouch 5 centimeters deep lined with mucosa. She had had discomfort at monthly intervals but of course no flow. A small mass apparently about 2 centimeters long could be felt by bimanual examination just above the upper limit of the vagina. No ovaries could be felt. Laparotomy was done at the request of the family to see if anything of a reconstructive nature could be done, although it was explained that this was quite unlikely. Exploration disclosed two small ovaries attached to the lateral walls of the pelvis, in one of which was a recent corpus luteum, passing downward from which were two retroperitoneal cordlike structures, the undeveloped muellerian ducts, which united in the median line to form a small fibrous mass about 2 by 1 centimeters in size, the undeveloped uterus.

My only purpose in this paper is to emphasize the frequency with which infections of the kidney may occur in women and to urge a more careful routine observation. Any woman in whose urine pus or blood cells appear is entitled to a careful effort to determine the source of the pus or blood and the nature of the infection, if any. An exception to this is the woman with a pyelitis of pregnancy in whom cystoscopy should be avoided if possible, but in whom it should be used if needed. The fact that within the past eight weeks four cases of tumor of the bladder have come under my own notice, would lend emphasis to this statement. One of these had been treated for ten weeks for intermittent hæmaturia. Cystoscopy disclosed an inoperable carcinoma of the vesicovaginal septum which is now under treatment by X-ray and radium.

CONCLUSION

1. Diagnosis is practically always possible if all means for arriving at a diagnosis be used.

2. Early recognition and treatment of a pyelitis may be the means of avoiding serious if not irreparable damage to the kidney.

MATERNAL MORTALITY, A CRIME OF TODAY[1]

By C. HENRY DAVIS, M.D., Milwaukee, Wisconsin

IN the past thirty years deaths from many diseases have been cut to a fraction of their former tolls. Between the years of 1890 and 1915 the death rate, per 100,000 population, for tuberculosis in the death registration area of the United States, fell from 252 to 145.8; pneumonia from 186.9 to 82.9; diphtheria and croup from 97.8 to 15.7; diarrhœa and enteritis under 2 years from 139.1 to 59.5; typhoid fever from 46.3 to 12.4.

The introduction of anæsthesia, aseptic methods, pre-operative treatment, and technical skill have made surgery so safe that surgeons think little of doing an exploratory laparotomy to make a diagnosis; and even some obstetricians deliver their patients by cæsarean section on the slightest indications.

During the past five years most of the world has been at war. In this war all the diabolical means of torture and death ever conceived of by the human mind have been to some extent employed. Millions of men have been wounded and sick. For the first time in history, battle has caused more deaths among our soldiers than disease. According to weekly bulletin No. 59 of the Chief Surgeon, S.O.S., of the A.E.F. "of the cases of disease: 90.2 per cent returned to duty, 6.0 per cent were invalided home, 3.3 per cent died in hospital, and 0.5 per cent deserted. Of the cases of traumatism: 73.8 per cent returned to duty, 21.1 per cent were invalided home, 5.7 per cent died in hospital, and 0.4 per cent deserted."

Yet in this day of progress and scientific medicine the act of becoming a mother remains as deadly for the average woman as it was before the discovery of anæsthesia and the introduction of antiseptic methods. The present maternal mortality is the greatest medical crime of today. A small group of physicians interested in obstetrics and the diseases of women have long appreciated that most of the maternal deaths are preventable. In their own work puerperal sepsis is a condition almost unknown except as it is seen in consultation. Very few deaths from the other complications of the puerperal state occur among the women they have carried through the entire period of pregnancy, labor, and the puerperium. The work of these physicians is ample proof that the present mortality is not due to the lack of scientific knowledge regarding obstetrical problems, but rather from failure to apply this knowledge. Women have always accepted the mortality and morbidity of childbirth as a sacrifice which they must lay on the altar of motherhood. This function of woman has always been cloaked with fatalism and guided by ignorance.

Between the ages of 15 and 45 childbirth is the second greatest cause of death among women. For the year 1915, in the registration area of the United States, there were, among women of these ages, 29,200 deaths from tuberculosis; 10,134 from childbirth, of which 4,173 were from puerperal sepsis; 8,766 from the various circulatory disturbances; 6,458 from all kinds of digestive disturbances; 5,549 from pneumonia, all types; 5,424 from cancer and other malignant tumors; while for these ages syphilis was reported as the cause of death 647 times and gonorrhœa 174 times.

Mortality statistics are always more or less inaccurate. Most writers believe the deaths from childbirth considerably greater than indicated by the above figures. However, we may assume that the errors will average up from year to year, and that similar errors will be made here and abroad. Dr. Meigs, in preparing her bulletin on maternal mortality for the Children's Bureau of the Department of Labor, studied the mortality records of sixteen countries. In deaths from puerperal sepsis the United States was fourteenth on the list. Only two, Switzerland and Spain, showed a higher death rate per 100,000 population.

It is unfortunate that the death rate from

[1] Read before the Chicago Gynecological Society, November 21, 1919. (For discussion, see p. 318.)

puerperal sepsis and other diseases caused by pregnancy and confinement has been estimated only per 100,000 men, women and children. The death rate per 1000 live births in the United States is only available for the year 1910 in the provisional birth-registration area and in 1915 and 1916 in the newly-established birth registration area.

In the provisional birth-registration area for the year 1910, one mother was lost for every 154 babies born alive. In Belgium for the same year the ratio was one maternal death for every 172 live births and in Spain one to every 175. Sweden on the other hand, had a record of one mother lost to every 430 live births.

Statistics show that grouping all women of child-bearing age together, tuberculosis alone is more deadly than childbirth. But if we leave out of consideration the ignorant foreign and tenement population among whom tuberculosis is so deadly, childbirth leaps to the front.

It is generally conceded that the type of man who carries life insurance with any of the old line companies, is representative of our best citizens. From the records of these companies we may hope to get some idea as to the relative frequency of death from different causes among the typical American citizens.

Through the courtesy of Dr. J. W. Fisher, medical director of the Northwestern Mutual Life Insurance Company, I have checked the family histories of 5000 applicants for life insurance. Both declined and accepted risks were included. These show that one man in every 17 who applied for insurance had a mother or sister or both who died from the immediate effects of childbirth; one in 27 from tuberculosis; and one in 47 from cancer or other malignant tumor. It is probable that a study of 100,000 applicants would modify this ratio to some extent. But checking the applications thousand by thousand as the series grew, it is found that childbirth always remained in the lead. Nor was there a great variation in the different years from which applications were studied.

I have long appreciated that our hospitals were constructed for the very poor and the well-to-do, but I was not prepared to believe that childbirth was so fatal for the mother and sisters of the average American citizen.

Have we as members of the Chicago Gynecological Society and teachers of medical students realized the present mortality from childbirth? If so, have we done our full duty toward bettering conditions in Chicago or elsewhere? Hospital beds for obstetrical patients are so scarce that less than 10 per cent of the women of Chicago can go to a hospital for confinement. The members of this society directly and indirectly through their hospital and dispensary services care for less than 10 per cent of the women delivered in Chicago. Nine-tenths of the women in Chicago and elsewhere are confined by midwives or physicians who are in the general practice of medicine or surgery, few of whom have had any special interest or training in obstetrical problems. Mortality statistics show that in 1916, 179 Chicago mothers died from puerperal sepsis and 220 from other puerperal affections.

Figures may be juggled, but the fact remains that the present maternal mortality is the greatest medical crime of today. However, destructive criticism is of itself useless. The profession may plead for leniency when charged with a crime of which it is not aware, but ignorance of the fact excuses no man if he has failed in his duty. It is the duty of every physician who undertakes to confine a woman to give her the best of his skill. Granting that the average physician has little skill in the management of an obstetrical case, he may at least try to give her a clean delivery. Much of the blame lies at the door of the medical colleges.

Obstetrics has always been the most exhausting branch of medicine and the poorest paid. That being the case, it was only natural that as the numerous commercial schools of medicine were developed it was difficult to persuade a doctor that the teaching of obstetrics would bring him a lucrative consultation practice. You are all familiar with the way those medical schools gave over a large part of their work to grandstand clinics in major surgery. Our medical colleges are still giving far too much time to the teaching of major surgical operations.

Due to the long-continued efforts of a small group of men interested in science of obstetrics the teaching of obstetrics is gradually being improved, but it is still the poorest taught subject in the average medical school. As an undergraduate at Rush I saw three normal deliveries. The graduate of today has seen at least twelve, and has usually had the opportunity to deliver one or more women under direction.

The man who goes into general practice, and a majority of our medical graduates do, must of necessity do more or less obstetrics. He must get his obstetrical knowledge as an undergraduate student or as a hospital interne. The average interne gets little or no obstetrical experience and goes into practice with only the superficial knowledge gained as an undergraduate. The present high maternal mortality is the logical result.

This unnecessary maternal mortality must be corrected, to a great extent, through the efforts of the specialists in obstetrics and gynecology. The dual specialty is and should remain a single specialty. The younger man entering it should do the harder, more demanding obstetrical phase while he is developing the mature judgment and technical skill required for the difficult obstetrical or gynecological operation. Later when less able to stand the strain of night work with broken sleep, he may take less of the normal obstetrics, turning it over to the young assistant, and reserve his strength for the cases requiring experience and operative skill. All the efforts to separate these specialties have retarded their progress, and, have turned hundreds of capable young assistants into practitioners of general surgery.

The American people must be informed regarding the dangers from the lack of surgical cleanliness and reasonable skill in the lying-in room. Hospital beds will not be forthcoming until women are educated to the fact that there is as much reason for going to a hospital to have a baby as there is for most of the surgical operations. Making the world safe for Democracy has been a popular slogan. However, most of us who wore the uniform were fighting to save our women from the horrors of an invasion. The war is over and the ravages of Belgium and France may soon be forgotten in the mad struggle for power and commercial supremacy. Already many of the people who claimed to be most bitter against the German are struggling for his trade. Will we who donned the uniform in a worthy cause return to our various hospitals and calmly ignore the crime at the door of our own profession?

INTRACRANIAL PRESSURE[1]

By CASSIUS C. ROGERS, A.M., M.D., F.A.C.S., Chicago

THE structures within the cranial cavity are normally just sufficient to fill it and nature has provided ways and means to maintain therein a relative constant pressure. Pathological conditions may so interfere with nature that a negative pressure or positive pressure exists. In either instance characteristic symptoms will be manifested. Often a slight negative pressure will produce more distressing symptoms than a relatively higher positive pressure. This is proved by the distressing headaches following the withdrawal of varied quantities of cerebrospinal fluid.

Either a negative or positive pressure may be produced by conditions from within or without the cranial cavity. The loss of cerebrospinal fluid or large quantities of blood are the most frequent cause of negative pressure. Even death has resulted from a too rapid withdrawal of spinal fluid, decreasing intracranial pressure.

It is my purpose to consider positive pressure in this paper.

The cranial cavity is bounded (except in infancy) by a firm non-elastic bony wall incapable of yielding except to great intracranial pressure. It is also surrounded by the second non-elastic structure, the dura, which yields but slowly to intracranial pressure and never rapidly enough to relieve acute conditions.

Positive pressure may be acute or chronic, the symptoms differing greatly with an equal amount of pressure. Of the acute the most frequent cause is perhaps injuries of the head with or without skull fractures. Intracranial lesions without fracture are often more serious than those accompanied by fracture.

A compound comminuted fracture without laceration of the dura produces within itself a decompression rendering extradural pressure impossible at the site of injury but intradural pressure may be but little relieved unless the dura is also severed. Cerebral hernia is always the result of increased intracranial pressure and no attempt should be made to reduce it by external pressure. If, however, the pressure becomes normal the hernia spontaneously disappears.

It must be remembered that intracranial hæmorrhage either extradural or intradural is never sufficient in quantity to kill, but it is the small hæmorrhage that would be considered trivial elsewhere, accompanied by œdema produced by irritation, that causes intracranial pressure sufficient to destroy life.

Acute extradural or subperiosteal pressures from hæmorrhage give symptoms that are pathognomonic and cannot be mistaken if closely observed. If the patient is unconscious, subjective symptoms cannot be obtained but if the pulse, temperature, respiration, and blood pressure are closely and frequently observed they will tell the exact condition of the patient and these alone will invariably tell, if properly interpreted, whether surgical relief is indicated. The acute pressure has a tendency to lower pulse, temperature and respiration, and increase in a direct ratio the blood pressure. The chart illustrates this.

If at the time of injury or hæmorrhage the patient is normal, if after a certain time the pulse is 60, temperature 98, respiration 16,

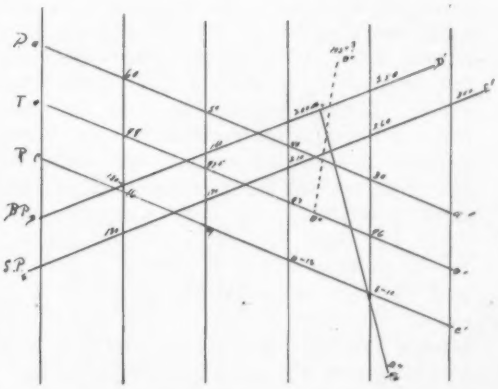

Chart showing acute extradural intracranial pressure. aa′, Pulse; bb′, temperature; cc′, respiration; dd′, blood pressure; ee′, spinal fluid pressure; D′D″, sudden drop in blood pressure; B″B″, sudden rise in temperature, at the same time a fall in blood pressure.

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blood pressure 130+, the findings should suggest at once that tihere s intracranial trouble which should demand close observation, preferably by a competent nurse. If the second observation in one or six hours, as the case may be, shows a steady decline—pulse 50, temperature 97, respiration 14, blood pressure 160+, — it is an indication that the pressure is increasing and if not surgically relieved disastrous results may follow.

Third observation—pulse 40, temperature 97, respiration 10–12, blood pressure 200+. A patient cannot live long with such findings. Sooner or later there will be a sudden change in the blood pressure. It drops suddenly to 90−, while the intracranial pressure is found by spinal puncture to remain high, even 200+ or 300+ millimeters of water. Now the time for operative interference has passed and death is the result with or without operation. Spinal fluid pressure remaining high with a sudden diminished blood pressure—(this broken relation)denotes disaster.

Acute extradural abscesses resulting from infections extending from the accessory sinuses, often give an entirely different picture as acute infections have a tendency to increase pulse, temperature, respiration, and often blood pressure. We have here two forces pulling in opposite directions and the result is that often the patient who has had an acute sinus infection with a rise in pulse, temperature, and respiration will suddenly have a normal pulse, temperature, and respiration, but apparently otherwise is much sicker than before. The sudden drop in pulse, temperature, and respiration apparently has been a common deceiver to some of our specialists in otology and has caused them to delay operative procedure, and brain abscesses and death has been the result. Experience has led me to believe that all cases of extradural and brain abscesses that are the result of sinusitis extension belong to the experienced general surgeon. As soon as a patient with acute sinusitis has an increase of pain associated with nausea and vomiting and slow cerebration, there is invariably an extension to the dura irrespective of the pulse, temperature, and respiration and radical surgical interference is demanded. If the infection has extended beyond the dura, localized or diffuse meningitis with the classic symptoms is present and lumbar drainage only tends to increase the area of infection.

In all extradural pressures, lumbar drainage for the relief of intracranial pressure is contra-indicated as only harm can be obtained. Lumbar puncture for diagnostic purposes is always permissible and advisable if done by those competent. If the infection is extradural the examination of the fluid may be negative.

Intradural pressure may be, subdural, subarachnoidal, intracerebral, and intraventricular. To relieve the pressure the dura must be opened, except in the presence of subarachnoid pressure, which may be relieved by lumbar spinal drainage.

The symptoms differ greatly as to location. Subarachnoid hæmorrhage may be associated with weak rapid pulse, slow irregular respiration with elevated, normal or subnormal temperature. Lumbar puncture reveals blood in the spinal fluid and while the patient is unconscious there are at times involuntary movements of groups of voluntary muscles, such as the moving of an arm or leg. The patients are restless and cry out at intervals. For this reason, the site of the lesion is sometimes looked for on the wrong side as the quiet side is taken for the paralyzed side while the irritation to the motor cortex by the blood produces contraction of the opposite arm or leg and the lesion appears to be located on the same side as the apparent paralysis which is never the case.

Acute intracerebral lesions located in silent areas produce symptoms very similar to the extradural lesions and cannot be relieved by lumbar drainage.

Acute intraventricular lesions or internal hydrocephalus cannot be relieved by lumbar drainage for either the foramen or Monro or Ilir or both are closed and the ventricles cannot be drained by drawing fluid from the subarachnoid space. Observations of the pupils and ophthalmoscopic examinations tell us little or nothing in acute cases but are always recommended.

There is one class of chronic intracranial pressure, however, that can be satisfactorily

treated by lumbar drainage. I have been unable to find mention of such cases in medical literature. If similar cases have been reported I would be glad to be informed where the reports can be found.

Recently I have been observing a series of cases that have in most respects a normal mentality. Some resort to petty thievery, others do many things that are contrary to a normal social standing, while others commit grave offenses. They apparently have a conscience but cannot control it. Attempts to discipline them have no lasting results. They may be punished and will immediately commit the same offense for which they were punished. They are bright and active, apt at learning and apparently are normal. Physical and roentgen examinations reveal nothing abnormal, questions are answered intelligently and correctly, until they think it is to their advantage to lie and then it is almost impossible to obtain the facts.

The pulse, temperature, respiration, and blood pressure are normal, there are none of the usual symptoms of intracranial pressure, no headache and no visual disturbances, but the spinal punctures reveal the cerebrospinal fluid under great pressure—from 200 to 300+ millimeters of water registered by the spinal fluid manometer. This may be our only clue that there is cerebral inefficiency. The rapidity of the flow of cerebral fluid from the needle gives absolutely no information as to the intracranial pressure. The pressure is easily reduced by lumbar drainage and remains low for days and perhaps permanently if proper treatment is administered. All laboratory findings are negative except the serological cerebral localization test of Abderhalden as done by Dr. Retinger at the Durand Hospital which invariably reveals in these cases a 2+ and often 3+ reaction of two or more of the glands of internal secretion: the pineal gland, pituitary (posterior), corporus mammilaris (anterior), 2+ or 3+ of the ovaries or testicles, suprarenals, thyroid, and frequently thymus.

The administration of the defective internal secretions will usually prevent the return of the increased intracranial pressure when it is once relieved by lumbar drainage.

As long as a normal intracranial pressure is maintained, by balancing internal secretions by supplying artificially the defective secretion, these patients remain good law-abiding citizens.

It is my opinion that many of our juvenile offenders belong to this class of cases and that our courts should sentence them to laboratories for examination instead of our present day reformatories or schools of crime.

Dr. B. O. Orndoff has furnished me with the following report on the examination of Miss B., one of a series of similar cases.

The examination consisted of roentgen observations of the head, thorax and abdomen, analysis of the blood, spinal fluid and urine together with a consideration of the history and physical findings.

The findings, which seem to have a bearing of importance in her case are in the head, a rather small pituitary fossa and some apparent disproportion of major anatomical structures which are probably developmental and not pathological. The chest is negative; the abdomen negative with the exception of some evidence of fixation of the cæcum and appendix, and the presence of a slightly enlarged ovary on the right side. Findings in the gastro-intestinal tract are negative other than those mentioned. The blood was found under normal pressure and gave a negative Wassermann reaction and no cytological proportions. The spinal fluid registered a pressure of 240 millimeters and decreased as low as 100 millimeters after the removal of 30 centimeters of fluid. The cell count of the fluid was negative as well as the Wassermann and Lange reaction. The physical findings were practically negative.

Dr. Retinger's serological report of Miss B.

The following are the results of the tests on the glands with the blood of Miss B.:

Liver	negative	Ovary	++
Adrenal	+++	Thymus	+
Thyroid	±	Spleen	+
Parathyroid	−	Lung	+
Kidney	−	Pineal gland	±
Pituitary (anterior)	−	Pancreas	−
Pituitary (posterior)	++	Placenta	−

It seems that if there is no brain trouble, there must be disturbance in the secretion of the adrenal and the ovaries. They are known to be in a close relation.

Candate nucleus	−	Ant. perforated space	−
Thalamus		Corp. mammillare	+++
Anterior nucleus	−	Geniculate Lat.	+
Median nucleus	± (+)	Superior Coll.	−
Lateral nucleus	−	Inferior Coll.	± (−)
Putamen nucleus	± (−)	Pineal	−
Optic chiasm	−	Pituitary (anterior)	−
Olfactory	−	Pituitary (posterior)	++
Dura	−	Pons	−
Fornix	−	Medulla	−
M. amygdala	−	Cerebellum	− all negative
Globus pallidus	−		

THE CLINICAL APPLICATION OF THE CARREL-DAKIN METHOD TO CASES OF ACUTE APPENDICITIS REQUIRING DRAINAGE

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UPON the well established foundation of clinical experience in dealing with the less advanced type of peritoneal infection rests the work of Depage in the treatment of certain war wounds. The so-called "abdominal technique" has been applied successfully to the closure of lung and joint wounds as well as extensive wounds of the soft parts after removal of foreign bodies or a resection of contaminated tissues. As in dealing with appendiceal or other intraperitoneal infections, the all-important point to decide has been the degree of contamination or infection. This recent confirmation of the great value of the discrimination between contamination and infection forces upon us in a new light certain considerations which had previously been accepted as a rather inevitable accompaniment of the course of cases of acute appendicitis requiring drainage.

From a review of 263 cases of appendicitis treated at the Presbyterian Hospital during the years 1915–1918,[1] we find that under methods of simple drainage there was a failure in a large percentage (38 per cent) to preserve in a satisfactory manner the very important tissues of the abdominal wall. These tissues at the time of operation are almost without exception free from infection. During the procedure of appendicectomy and drainage of a peritoneal abscess an extensive contamination of the operative wound necessarily occurs. The time has arrived to consider the possibility of preventing this contamination from proceeding unchecked to the stage of destructive infection. It was found in the cases reviewed that the average period of suppuration was 15.4 days, during which time the majority of the wounds discharged foul pus and sloughs. The average duration of hospital stay was 28 days. Among the frequent complications, fæcal fistula developed in 7.5 per cent of the cases. There was a 9.1 per cent mortality. Whether the course of these cases may be improved in any of these essential particulars by the use of antiseptics is dependent upon their safe application and a determination of their efficiency in this type of infection.

As a result of the experimental study of certain chlorine antiseptics in localized peritoneal infections in dogs[2], the antiseptic treatment of cases of acute appendicitis requiring drainage was undertaken first in February, 1918, as a routine in the wards of the Surgical Service at the Presbyterian Hospital.

Aside from the eighteen cases which have been treated by the writer and which form the basis of this report, the method has been employed by others in 13 cases of appendicitis and in a number of intra-abdominal drainage tracts accompanying other conditions.

Before attempting the application of Dakin's fluid to appendiceal infections, the following conclusions had been reached in regard to the considerations involved:

1. Suture material in the intestinal wall apparently is not materially affected by small, repeated injections of Dakin's fluid.

2. Fibrin deposits occur in the presence of Dakin's fluid; adhesions walling-off intra-abdominal drainage tracts may remain effective, and healing of intestinal wounds may occur after repeated injections over a period of several days.

3. Severe pain reactions and shock attend the introduction of Dakin's fluid into the free peritoneal cavity. The use of this antiseptic must, therefore, be restricted to the treatment of the drainage tracts after the period of walling-off has occurred. The injections are, therefore, not intraperitoneal but intra-abdominal.

[1]Rulison, E. T. Jr. Drainage in appendicitis. A review of cases of acute appendicitis requiring drainage, at the Presbyterian Hospital, 1915–1918. Ann. of Surg., 1919, Dec.

[2]Rulison, E. T. Jr. A study of the intra-abdominal use of Dakin's fluid and dichloramine-T solutions with special reference to local peritoneal infections.

4. The satisfactory establishment of a water-tight drainage tract seems to depend as to its rapidity of formation upon the type of infection. The structure of the drainage tract in certain severe infections may be very precarious, even after a considerable interval. Even in the milder infections, a minimum of adventitious tissue is often present between adherent coils. Great caution as to time and manner of introducing the fluid is, therefore, necessitated. Accidents involving the integrity of intra-abdominal drainage tracts, with escape of Dakin's fluid into the free peritoneal cavity, are attended by grave danger, especially if infection be present.

5. Less slough and less foul discharge were noted in the cases treated by Dakin's fluid.

Finally, in some localized peritoneal infections in dogs, Dakin's fluid may be used with comparative safety, provided:

1. Sufficient time is allowed to elapse for a water-tight drainage tract to form;

2. Mechanical pressure of tubes or fluid upon the walls of the tract is avoided.

With these considerations in mind, the antiseptic postoperative treatment was undertaken. At first, Dakin's fluid of half strength was used and the introduction of the solution intra-abdominally was delayed for several days in order that there might be no question as to the establishment of a proper tract. In the later cases, having gained more confidence in the method, full strength Dakin's fluid has been used superficially in the planes of the abdominal wall from the time of operation and after 48 to 60 hours in the deep abdominal drainage tracts.

Attention in the 18 cases reported has been directed especially to the course of wound infection, noting the possible influence of the antiseptic in preserving the abdominal wall tissues, and in altering the course of intra-abdominal suppuration. The bacteriology of the wounds has been carefully studied, especially from the viewpoint of determining whether or not secondary suture of the wounds were possible or feasible. Many cases have in this way received injections for many days after gross suppuration has ceased.

The contrast to the cases treated by simple drainage or by daily irrigation has been sharp, the period of discharge of foul smelling pus with shreds of slough from abdominal wall or abscess cavity in all cases being greatly lessened and in some cases minimized to a striking degree. Although the group is one of fully average severity there have been surprisingly few complications. There have been no fæcal fistulæ. No serious accidents have attended the use of Dakin's solution intra-abdominally, nor have the patients suffered pain reactions following injections, except two late injections in one case. The patients have been discharged with better wounds and the follow-up results to date indicate that the percentages of herniæ will be materially decreased.

DESCRIPTION OF THE METHOD

No essential change has been made in the operative technique of the cases. When possible the stump of appendix has been inverted and the site closed by a purse-string of Pagenstecher, reinforced by a figure-of-eight suture of plain catgut. In the cases with cæcal œdema precluding this treatment, the stump has been crushed and ligated with chromic catgut.

Type and arrangement of drainage tubes. A straight drainage tract is essential. We have not attempted the Dakinization of a curved or tortuous sinus and feel that any attempt to do so would be fraught with danger. A straight tract may always be obtained, e.g., in draining the right lumbar gutter, by insertion of the drains through a stab wound in the loin. In some of our cases with straight and tortuous tracts we have treated one tract with Dakin's fluid and the other by daily irrigations of saline solution.

The selection of the rubber drainage tube is important. We are using a modification of the double arm tube devised by Dr. Joseph A. Blake. The tube is of soft rubber having a wall 2 millimeters in thickness and a lumen of 6 millimeters. Instead of fashioning the tube so that the deep ends of its two arms are held together by a portion of the tube wall, two separate tubes are taken and their beveled deep ends are held together by an

untied loop of plain catgut. One tube is fenestrated by means of a harness punch, the fenestrations being 3 to 4 millimeters in diameter and placed in alternating axes 2 centimeters apart. A strip of narrow gauze packing moistened with 5 per cent dichloramine-T in chloracosane is placed in the fenestrated tube to prevent possible insinuation of a process of momentum, to provide capillarity between the deep end of the tube and the dressings, and for its germicidal effect upon the organisms in the discharge (Fig. 1, A). A long bevel to the deep end of the tubes is desirable permitting a readier collapse of the walls after removal of drain, and lessening the possibility of direct pressure on intestinal walls which are in contact. The non-fenestrated or closed arm is used principally for the purpose of obtaining a larger drainage tract; it also provides freer drainage during the first 48 hours. The drainage tubes must be long enough to project an inch beyond the skin, so that they may discharge into dressings superficial to a thick non-absorbent pad (to be described).

When two drainage tracts are desirable, the double tube may be used in each or a single fenestrated tube with wick used in the shorter tract. We have also employed the single fenestrated tube alone in cases with short tracts (Fig. 3).

Great care should be employed in placing the tubes to obtain a straight drainage tract. This injunction holds good whether antiseptics are to be used or not, but is absolutely essential to the safe employment of Dakin's fluid.

Closure of the abdominal wall. The peritoneum is closed about the tubes with continuous plain catgut. A tight closure is impossible because of the triangular spaces formed by the walls of the tubes and the edge of the peritoneum. These triangular spaces are closed by the introduction of narrow strips of vaseline gauze, the outer ends of which lie upon the skin surface.

The contaminated planes of the abdominal wound are now thoroughly sponged with Dakin's fluid and the transversalis and internal oblique muscles then loosely united about tubes with interrupted plain catgut. The aponeurosis of the external oblique is then closed loosely about the tubes by continuous or interrupted plain catgut, and the skin by angle sutures of silkworm-gut. Untied sutures of silkworm-gut may be placed near the center of the wound.

The skin is now lifted by a small retractor and a 5 centimeter Carrel tube (Fig. 1, D) inserted toward the angle of the wound so that it may lie along the sutured aponeurosis. A second Carrel tube is placed along the aponeurosis toward the opposite angle of the wound. If the wound has been closed in such a manner that the drainage tubes emerge at one angle, but a single Carrel tube, of course, is required.

Separation type of dressing. The employment of the closure just described together with the separation type of dressing is an attempt to prevent the contamination of the planes of the abdominal wall from resulting in an infection and consequent tissue destruction. With this idea in view vaseline strips are placed upon the skin surface, in accordance with the Carrel-Dakin technique, a few gauze compresses and then a Carrel pad, consisting of a layer of absorbent cotton toward the wound and a layer of non-absorbent cotton superficially. The drainage tubes are permitted to extend through this pad and discharge their infectious material into gauze compresses placed externally. The ends of drainage tubes are transfixed by safety pins (Figs. 2 and 5).

The Carrel tubes to the aponeurosis on emerging from the wound lie flat along the skin surface and are held by a strip of adhesive to the skin of the upper abdomen. The ends of these tubes are kept covered by a gauze compress (Fig. 3).

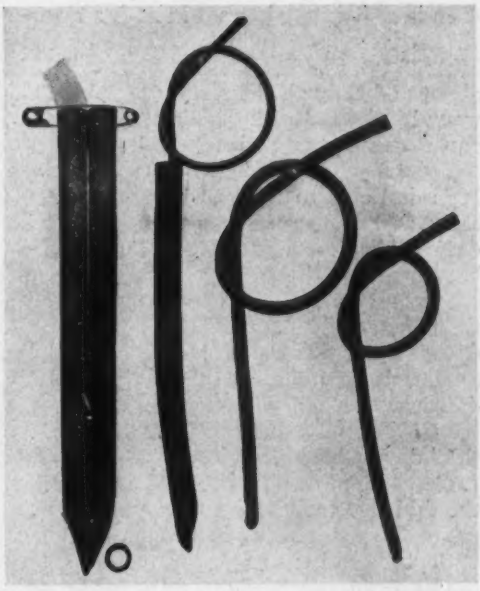
Five cubic centimeters of full strength Dakin's fluid is injected through each of the Carrel tubes before the patient leaves the table, and a similar amount is ordered to be given every two hours night and day.

That the separation type of dressing efficiently functions during the period of its employment (48 to 60 hours) has been repeatedly demonstrated. Foul exudate has been found in the superficial dressings and thin serous exudate with a chemical odor

saturating the dressings in contact with the wound. During this period it would seem that the various re-entrant angles of the wound are permitted to fill with clean fibrin and organization may be well on its way before exudate from the deep tract is permitted to come in contact with the wound.

Postoperative care of wound. First dressing. If operation has been at night the first dressing is done on the following day at 12 to 18 hours; if a morning operation, the first dressing is at 24 hours. All dressings are carefully removed, clean forceps being used in handling dressings beneath the Carrel pad. Fresh compresses and pad are applied. The dichloramine strip is removed from fenestrated tube after application of the fresh pad and smears may be taken from its deep end. A fresh wick of 5 per cent dichloramine-T is introduced into the same tube and 1 to 2 cubic centimeters of the same solution dropped into the closed tube.

Second dressing. If this comes at 36 hours the procedure of first dressing is repeated in every detail. If the second dressing comes at 48 to 60 hours all wound dressings are removed including vaseline strips to peritoneum and the closed drainage tube is also removed. This is best accomplished by grasping it with an artery clamp and loosening it first by a twisting motion, thereby freeing connection by catgut loop to the fenestrated tube. Following its removal the fenestrated tube is loosened and withdrawn about 1 centimeter. It is now trimmed off close to the skin, as separation dressings are no longer used, the deep and superficial wounds freely communicating. Unless there is reason to fear that adhesions have not formed about the drainage tubes (possible in certain cases of acute diffuse peritonitis), a trial injection of saline is made by a small (No. 12 French) soft rubber catheter placed to the depth of the fenestrated tube. With a straight drainage tract, the solution should promptly return *both around and within the tube.* (Fig. 1, B.) Five cubic centimeters of Dakin's fluid warmed to 100°F. is now gently injected. A 10 cubic centimeter Luer syringe is very satisfactory as the fluid is accurately measured and the weight of the plunger is sufficient to



A *A*[1] *B* *C* *D*

Fig. 1. *A*, Modified Blake tube employed at operation for deep abdominal drainage. *A*[1] Cross section of tubing. *B*, Fenestrated arm of modified Blake tube, shortened at outer end and a No. 12 French catheter introduced through lumen; arrangement employed when antiseptic is first instilled in deep abdominal drainage tracts. *C*, No. 18 French catheter employed after removal of fenestrated tube for instillation of Dakin's fluid into abdominal drainage tract. *D*, Carrel tube (5 centimeter type) used for introduction of Dakin's fluid along line of sutured aponeurosis.

cause fluid to run in. If the patient gives the slightest evidence of pain reaction, several hours to a day should be allowed to elapse before again attempting injection. Pain reaction means that the tract is not yet watertight; it means that Dakin's fluid is reaching peritoneum not protected by fibrin. In none of the 18 cases reported have we had a pain reaction at this time. Dakin's fluid warmed to 100° F. is ordered to be injected slowly and gently in 5 cubic centimeter amounts every two hours, all treatment to be discontinued immediately if patient develops a pain reaction. The instillation catheter is left in place, the outer end being held by adhesive strip to skin along with the Carrel tubes. Thus at 48 to 60 hours we may introduce full strength Dakin's fluid (Fig. 6)

even in a deep pelvic tract in such a manner that it may come fully in contact with the infected tract wall. The fluid has exit both about and through the remaining tube. The wound is dressed with vaseline gauze to skin, gauze compresses, and a Carrel pad.

Third dressing and subsequent dressings. It is desirable to remove drainage tubes as early as commensurate with safety. With deep pelvic tracts and severe infections we have maintained the above arrangement and continued the injections by catheter within the fenestrated tube until the fourth or fifth day. In some cases we have substituted a smaller sized fenestrated tube at the third day dressing. When dealing with a short tract, a No. 18 French catheter is introduced on the third day and fenestrated tube removed (Fig. 1, c). This is easily done with no injury to the drainage tract and no discomfort to the patient by introducing it through the lumen of the tube and slipping the latter out over it.

The arrangement of catheters in the deep tracts with Carrel tubes still in the superficial wound is the final one (Fig. 7). No further changes are made until the wound is clinically clean. The dressings from the third or fourth day are extremely simple. The amount of Dakin's fluid intra-abdominally is usually increased rapidly to 10 cubic centimeters. More might be used with safety and perhaps with benefit if infection does not seem to be held in check. We have found that even in a long tract 10 cubic centimeters is amply sufficient to flush it, and that with the 10 cubic centimeters injected superficially the dressings do not become too saturated.

The Carrel tubes are usually forced out by the healing tendency of the wound about the seventh to tenth days. It is not necessary to disturb them. They may be removed, if still in place, as soon as discharge has become negligible.

Sutures are removed at the end of a week. As a rule the reaction about sutures is slight, in many cases not more than is found in a wound healing by primary union. If untied sutures have been placed at operation they may be tied when appearance of wound and bacterial count justifies.

Smaller catheters may be substituted at any time when there is indication that the tract is closing down too tightly about the original instillation catheter. In most cases we have observed this precaution about the eighth or ninth day. The only accident accompanying injection in our cases occurred on the thirteenth day in a deep pelvic tract which fortunately had been clean for at least 48 hours. The walls of the tract had closed down about the instillation catheter and the injection of 10 cubic centimeters of Dakin's was given with perhaps a little more force than usual. The patient suffered a sudden sharp pain in the lower abdomen which continued for over an hour. An injection two hours later failed to return, indicating that the fluid was entering the free peritoneal cavity. The catheter was removed, all treatment stopped, and the patient made a speedy recovery.

Daily bacterial counts have been made in all the cases. While this is not an essential and while the scientific accuracy of the counts may be questioned, we have found them of some practical value. The drop from infinity of organisms usually corresponds closely with the stage of clinical cleanliness when drainage may safely be discontinued. The counts, therefore, aid in a nicer determination of the condition of the wound.

When possible the dressings are done just before a Dakin's injection is due and smears are made either from the deep ends of the wicks in the early dressings or from exudate pressed gently from the wound. Smears from the superficial wound during period of separation dressings have been made and recorded in several cases. These of course are of purely academic interest. Smears taken and recorded for long periods after suppuration had ceased, to determine possibility of safe secondary closure did not indicate that this procedure is feasible.

Treatment of wound after peak of infection has been passed. That secondary closure by suture in the average case is neither feasible nor safe has been a disappointment, but can be satisfactorily explained by the biologic and dynamic conditions present. An abdominal drainage tract represents a dead space, the walls of which as infection declines are lined by granulation tissue. These walls have a very

decided tendency to collapse and obliterate the dead space. In the re-entrant angles of this collapsing cavity micro-organisms may remain in pockets. Sudden increases in the bacterial counts after negative counts for several days indicate the presence of these pockets. While it is theoretically possible that by continued injections of Dakin's fluid the infected cavity might eventually approach nearly enough a sterile condition to permit successful closure of the skin, the time element involved renders such attempts apparently futile. There is a great mass of clinical evidence to prove that residual abscesses rarely occur when drainage is discontinued after suppuration has declined and the wound appears clean. The injection of any fluid, antiseptic or otherwise, into the tract after healing has begun directly opposes the major factor of the healing tendency, i.e., collapse of the surrounding walls. The remaining factors in healing, the shrinkage of tissue and proliferation of new tissue are minor considerations.

In our last cases we removed the instillation catheters and Carrel tubes (if they still remained) just as soon as the wound ceased to show signs of suppuration and bright coral red granulations were in evidence, and applied a dry dressing. The bacterial counts in all these cases showed at this time few if any organisms. All of these cases left the hospital before the twentieth day with wounds healed or with small granulating areas flush with skin surface.

The reader is referred to the case reports and bacteriological charts for detailed description of the technique used under varying conditions.

Tabulation of the 18 cases gives the following data: acute appendicitis with acute local peritonitis was the final diagnosis in 6 cases, *acute appendicitis with abscess in 11 cases*, and acute appendicitis with peritoneal abscess and acute diffuse peritonitis in 1 case.

Intermuscular incision was used in 8 cases, intermuscular with Weir extension in 7 cases, and right rectus incision with splitting of muscle in 3 cases.

The lesions of the appendix found were: acute inflammation with fibrin on surface in 4 cases; gangrenous appendix in 14 cases; perforation of appendix had occurred in 7 cases. Thrombosis of the meso-appendix was noted in 1 case and gangrenous omentum in 1 case.

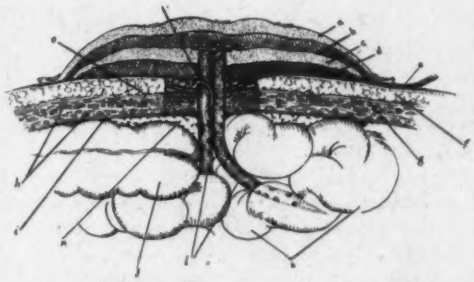


Fig. 2. Diagram illustrating arrangement of tubes and dressings at time of operation. *a*, Pad of absorbent cotton. *b*, Gauze compress layer (outer). *c*, Pad of non-absorbent cotton. *d*, Gauze compress layer (inner). *e*, Carrel 5 centimeter tube to suture line in aponeurosis. *f*, Panniculus. *g*, Aponeurosis of external oblique muscle. *h*, Muscle planes. *i*, Parietal peritoneum. *j*, Cæcum. *k*, Intestinal coils. *l*, Fenestrated soft rubber drainage tubes (closed tube to pelvis not indicated). Fenestrated tube appears too angulated. *m*, Strip of gauze packing placed in angles between tubes. *n*, Omentum. *o*, Sutures.

Free clear exudate was found in 1 case; free cloudy exudate without odor in 6 cases; free foul pus in 2 cases. Localized pus collections were present in 12 cases, in 3 of which the amount exceeded 100 cubic centimeters. Multiple pus pockets were present in 1 case. Foul pus at operation was found in 12 cases. The location of abscesses was as follows: pelvic, 5; subcæcal, 2; procæcal, 1; mesiocæcal, 1; retrocæcal, 1; extraperitoneal, 3 (retrocolic, 2; retrocæcal, 1).

The appendix was removed completely in 16 cases, in 14 of which the stump was inverted and site re-inforced in 13 cases. In 2 cases the stump was crushed and tied off. In one case the appendix had sloughed off near its base and the necrosed portion which contained fæcalith was alone removed.

Cultures were taken at operation in all cases. There were no sterile cultures reported. In 2 cases, however, final culture report was not recorded but in each of these cases organisms were present on smears taken at operation. In the 16 culture reports bacillus coli communis was present 12 times; bacillus coli communior, 1; streptococcus hæmolyticus,

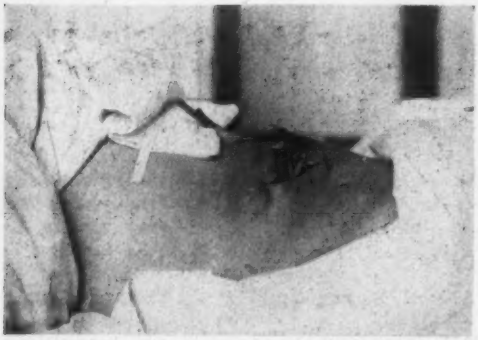


Fig. 3. Appearance of wound at the first dressing. This case was one with a short, straight tract and one fenestrated tube was used. There are two untied sutures near the center of the wound. Carrel tubes are seen entering and pass along the surface of aponeurosis toward either angle of the wound. Their free ends are held by adhesive and the tips are covered by gauze.

1; streptococcus non-hæmolyticus, 2; streptococcus viridans, 1; streptococcus not differentiated, 5; bacillus pyocyaneus, 1; gram negative bacilli, 2.

Double rubber tube drains were used in 11 cases, in one of the cases two sets being inserted and in 5 of the cases an additional single fenestrated tube was introduced. In 4 cases single rubber tubes were used, for the most part cases with abscesses near the operation wound. In 3 cases multiple single tubes were used. No cigarette drains were employed. Drains were placed to pelvis 13 times, to cæcal region 12 times, and to right lumbar gutter 2 times. All drains were introduced through the operative wound.

Closure of peritoneum in a manner to preclude continued contamination of the wound from peritoneal exudate was undertaken in 16 cases. Separation type of dressings was applied in these cases. The only cases in which this part of the technique was not employed were the first case of the series and one case in which particularly free drainage was thought desirable because of the widespread infection present.

Skin closure was loose in 17 cases. We had cause to regret the snug closure in the remaining case. The aponeurosis of the external oblique was included in the skin suture in 3 cases, 2 of which were ill advised. The one case in which the procedure was perhaps justified was one with a long right rectus incision and with marked distention of intestines. Untied sutures were placed in skin near the center of the wound in 4 cases.

Carrel tubes were placed along the aponeurosis in all cases.

RÉSUMÉ OF POSTOPERATIVE WOUND TREATMENT

Early replacement by smaller drains, or removal of the closed tube arm was carried out in all cases, the average time for this maneuver being 2.6 days.

The average time at which instillation catheters alone were present in the drainage tract was 4.5 days.

Of the 27 tracts present in these 18 cases all but 4 were treated by Dakin's fluid. Two of these were tortuous and in 2 the drains were removed early without replacement.

Dakin's fluid was used on an average of 9 days in the superficial wounds, the average amount being 10 cubic centimeters every two hours.

The average time at which Dakin's was first introduced into the deep drainage tract was 2.7 days (65 hours) from time of operation. The average time when Dakinization of the deep tracts was discontinued was 13.6 days after operation. The average duration of the deep antiseptic injections was, therefore, 11.2 days. The amounts of Dakin's fluid used varied from 2 to 10 cubic centimeters. In all cases except the first, 2 full strength Dakin's was used and 5 to 10 cubic centimeters in each tract was the customary amount.

Antiseptic treatment of the deep tracts was abruptly discontinued because of complication in 3 cases: the first (Case 3) on the tenth day because of indications of residual abscess; the second (Case 7) because of mechanical ileus; the third (Case 9) because of accident to drainage tract during injection on the thirteenth day. *Vide supra.*

Five per cent dichloramine-T was used at operation and during the first 48 hours in 15 of the cases.

There was evidence of insufficient drainage in but one case (Case 3). On the tenth

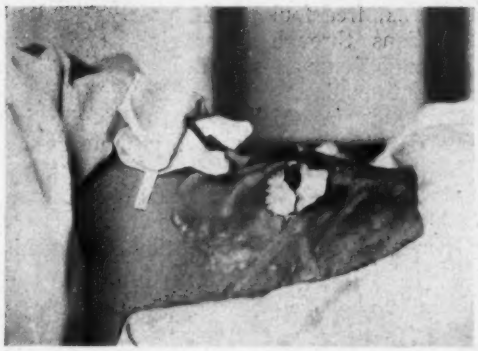


Fig. 4. The same wound as in Figure 3 with vaseline strips laid on the skin surface and a single gauze compress placed about the tube.

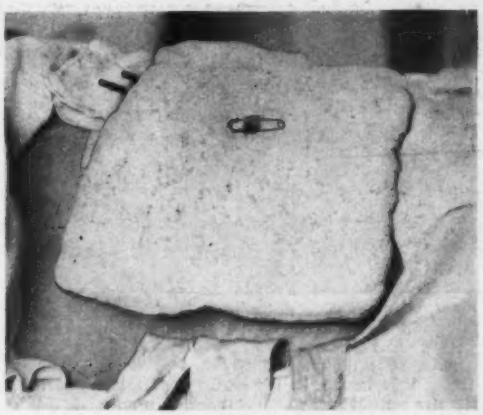


Fig. 5. The same as Figures 3 and 4, with Carrel pad applied. The drainage tube is allowed to project through this pad of absorbent and non-absorbent cotton and discharges into the superficial gauze compresses which are applied and held by the taped adhesive straps which may be seen turned back.

day signs and symptoms of residual abscess developed. Dakin's injections were discontinued. Measures to relieve condition were unnecessary as the wound shortly spontaneously discharged a quantity of exudate.

The re-institution of drainage was not necessary in any of the cases nor were measures to improve drainage necessary.

The average time when all treatment was stopped and dry dressings applied was 13.8 days.

There was no gross suppuration present in one case, slight suppuration in 5, moderate suppuration in 7, and profuse in 5.

The discharge was odorless throughout in 2 cases. The average duration of odoriferous discharge in 15 cases was only 6 days, and in a number of cases the odor was slight.

Seven cases passed through their period of drainage without apparent slough; in 8 cases the slough was superficial or from the deep tract (abscess wall); in 3 cases only was there definite slough of aponeurosis, and in 2 of these the slough was what would ordinarily be considered slight. The one case in which the aponeurosis suffered considerable damage was Case 7. In this case the aponeurosis was included in the skin sutures and Dakinizatson of the superficial wound was thereby rendered inefficient.

There were no cases of disruption of the wound, and but one (Case 7) in which real wound infection occurred. In no case did the intestine herniate into the wound.

Bacterial counts ranged below 60 per field throughout in 4 cases. In 13 cases the counts remained at infinity until the eighth day (average) when a drop in the number of bacteria occurred. The earliest drop from infinity was the third day and the latest drop the twelfth day. The drop was rapid and abrupt in some cases and slow in others.

The average time when suppuration ceased and wounds were entirely free of slough and lined by bright red granulations, with bacterial counts low was 10.9 days (16 cases treated throughout by antiseptics). The 2 cases excluded are Case 7, with mechanical ileus interrupting treatment on sixth day, and Case 12, death on fourth day.

Closure of wound by suture was attempted in 5 cases with 3 successes and 2 failures. Gradual closure aided by strapping with adhesive has been the rule, and has proved the most safe and practical method in our hands.

Certain complications were in a way anticipated, as we were proceeding in an untried field. While the early introduction of Dakin's fluid in the local peritoneal infections of the dog gave us a certain degree of assurance, the reaction of the human peritoneum was an unknown factor that could only be determined by careful trial. That Dakin's

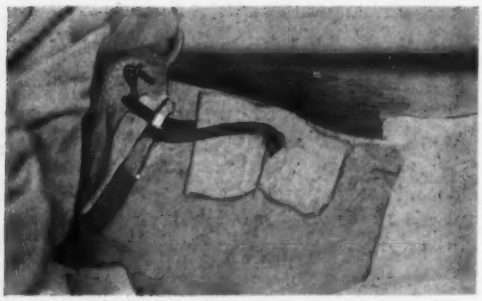


Fig. 6. Case having two drainage tracts (cæcal and pelvic). Arrangement of tubes at end of 48 hours. The closed tube to the pelvis has been removed and the fenestrated tube loosened and shortened. Trial injection by catheter seen entering end of tube has been made. The cæcal tube (one only) has been loosened and shortened and an instillation catheter introduced. Carrel tubes to superficial wound appear emerging from gauze compress.

fluid did not destroy protective adhesions, that suture material imbedded in tissue was not acted upon in any serious manner, that secondary hæmorrhage and fistula did not result from its use was very soon established. In these 18 cases there has not been a single case of secondary hæmorrhage or fæcal fistula. There has been but one residual abscess which emptied itself spontaneously through the drainage tract.

The only serious complication of the series was a mechanical ileus, which was probably due to tube pressure. In this case a single tube of unusually large lumen and very thin wall was placed to the pelvic abscess. The end of the tube was not properly beveled, and to prevent possible buckling the lumen was filled out with more gauze than is advisable. At operation on the sixth day an angulated loop was found in the pelvis in a position indicating possible pressure from the drainage tube. The condition of the tract was found unusually clean for the sixth day and the case made an uninterrupted recovery. Dakin's was not used after the operation for relief of ileus and there was considerable wound suppuration, resulting in a ventral hernia.

There were two pulmonary complications, one an acute bronchitis, the other a probable pulmonary embolism occurring in the one fatal case. The patient (Case 12) was a very obese woman, age 50, with a gangrenous perforated appendix, free foul pus in poorly localized collections through lower right abdomen-visceral peritoneum covered by thick fibrino, purulent exudate. The omentum fully 5 centimeters in thickness was acutely inflamed. That her resistance was unusually low was indicated by a low leucocyte reaction after more than 48 hours of infection. (White blood cells 7,400, polymorphonuclears 78 per cent.) Following operation her abdominal condition was apparently improving, when on the fourth day she became dyspnœic, cyanotic, face flushed, and heart action irregular. Her cough was unproductive. Fine moist crepitant râles were heard at both bases. Death occurred 102 hours after operation. Unfortunately no autopsy was obtained. In this case separation dressings were not employed. Dakin's fluid was introduced in the deep tracts after 48 hours, with free return and no pain reaction.

The condition of wounds on discharge was as follows:

Healed 2 cases; healed with clean crust 2; small granulating areas flush with skin surface 6; large granulating area (0.5 by 2 centimeters) 1; with shallow sinus 6. There was only one case discharged with a weak wall (Case 7 Dakin's treatment interrupted by development of mechanical ileus).

The average duration of hospital stay was 24.1 days. During the developmental period of the technique, several cases were Dakinized for longer periods than routine practice would demand.

Sixteen of the 17 recovered cases have been followed up over a period of 2 to 6 months. There have been no symptomatic failures indicating untoward late peritoneal changes (adhesions) and no economic failures. Unfortunately the exact time of resuming work or play has been recorded in but 3 cases. One of these was a child who resumed full play on reaching home, the other 2 resumed work at the end of 3 weeks after leaving hospital. The anatomical results were excellent in 12 cases (75 per cent); there was a slight bulge present in one case (rectus incision) at 6 months and a diffuse bulge at 2 months in the child referred to above; there were 2 hernias (12.5 per cent), one small

and one large, the latter occurring in case not Dakinized throughout.

There have been no late secondary complications or operations.

While 18 cases represent a small series it comprises infections of fully average severity and the results show a decided improvement in many respects over those attained under simple drainage treatment, when comparison is made either with the 263 cases reviewed[1] as a whole or the cases handled by any one of the various methods employed by individual operators (Table I).

Dakin's fluid and dichloramine-T solution have been used by several surgeons at the Presbyterian Hospital in the treatment of their cases during the past few months. In this early period of the use of antiseptics intra-abdominally we have all endeavored by various means to render the method of their use as simple as possible. The author is indebted for many valuable suggestions to the experience of other members of the staff who have employed Dakin's fluid in the treatment of their cases. A review of the 13 other cases of appendicitis in which antiseptics were employed shows no serious complication attributable to the method. Fæcal fistula occurred in 2 cases, one thought to be due to prolonged pressure of the drainage tube; the other occurred at the twentieth day. Hæmorrhage occurred from a right rectus wound in one case. Two secondary abscesses developed, one mural and one peritoneal. There was one death. The average duration of hospital stay was 24.1 days. As most of the cases have been recent ones the follow-up results are incomplete.

Fig. 7. Same as Figure 6, with cæcal and pelvic tracts. The drainage tubes have been removed leaving No. 18 French catheters in the two tracts. One of the Carrel tubes has been forced out by rapid closure of the upper angle of the wound. Untied sutures may be seen knotted and strapped back.

The following observations have been made in the cases treated by others when favorable expectations were not being realized:

1. That Dakin's fluid was not begun in the superficial wound until the third or fourth day.
2. That insufficient amounts of Dakin's fluid were used (1 to 2 cubic centimeters) both superficial and deep.
3. That injections have been given at infrequent intervals, every 3 to 4 hours,

[1] Drainage in appendicitis. Ann. Surg., 1919, Dec.

TABLE I

	263 cases reviewed	Best results attained under methods of simple drainage			Antiseptic treatment Author's 18 cases
		A_1 (30 cases)	B_2 (28 cases)	C_3 (29 cases)	
Period of suppuration	15.4 days	13.5 days	15.6 days	13.8 days	10.9 days
Immediate secondary procedures (to improve drainage, etc.)	16.4 per cent	3.3 per cent	10 per cent	20 per cent	5.5 per cent
Fæcal fistula	7.5 per cent	10 per cent	0 per cent	6.9 per cent †	0 per cent
Duration of hospital stay (initial)	26.5 days	22.7 days	23.4 days	29.9 days†	24.1 days
Perfect anatomic, symptomatic, economic results	58 per cent —*	53.4 per cent	57.1 per cent	63.7 per cent	75 per cent
Weak walls	per cent per cent	per cent per cent	per cent per cent	per cent per cent	per cent per cent
Bulges	18	20	14.3	22.7	12.5
Hernia	20 — 38	26.6 46.6	28.6 42.9	13.6 36.3	12.5 25
Deaths	9.1 per cent	3.3 per cent	3.6 per cent	3.4 per cent	5.5 per cent

A_1—Cases were treated by cigarette drains, later removal, few irrigations.
B_2—Cases were treated by early replacement of tube drains by catheter and daily irrigation.
C_3—Cases were treated by early loosening and shortening of tube drains, with irrigation, late replacement of drains by smaller tubes.

* In 4 per cent condition of abdominal wall could not be determined from the records.

†There were 5 secondary procedures in one case, making total unusually high; 2 fæcal fistulæ in 1 case; one case remained in hospital 205 days. Average duration of hospital stay with this case excluded-23.2 days.

or discontinued entirely while patient was sleeping.

4. That drainage tubes have been left *in situ* for 5 to 7 days and Dakin's fluid given only within the tubes allowing little or no contact of antiseptic with wall of tract.

5. That Carrel tubes for protection of aponeurosis have been improperly placed:

a. Wound sutured too tightly about these tubes, making injection difficult and forcible injection causing dissecting up of the loose tissues.

b. Tubes introduced from the angles of the wound rather than the center, with gaping of wound resulting after a few days.

While unremitting care and attention to essential details are absolutely necessary in the antiseptic treatment of these cases, it is the author's conclusion that Dakin's fluid may be used with safety and with far better immediate and probably better remote results than may be attained by simple drainage.

While it is not within the scope of this report to enter into a discussion of the exact manner in which a 0.5 per cent neutral sodium hypochlorite solution may act as an antiseptic, certain clinical observations and perhaps speculations may be of interest. It does not seem too much to hope that with the proper introduction in adequate amounts of this or some other antiseptic the vitally important tissues of the abdominal wall may be completely preserved in all cases by preventing the stage of contamination from passing to one of infection. This, in the author's opinion, is the major part of our problem. The abdominal drainage tract in these cases within 48 to 72 hours represents a limited infected dead space, the walls of which are lined by fibrin, leucocytes (dead and living) and bacteria. The structure of the walls of the tract is in all cases a delicate one and as the coils of intestine may be united by minimal adhesions, gentle treatment is entirely essential if antiseptics are to be introduced. The tensile strength of the drainage tract as treatment continues, probably decreases rather than increases. The introduction of an antiseptic having the known irritant properties of Dakin's fluid within the general abdominal cavity must always be done under low pressure and with certain and unimpeded return into the wound dressings. While the degree of infection in the dead space of the drainage tract must increase rapidly during the first 48 to 60 hours after operation, delay of introduction of this antiseptic during this period seems necessary from the viewpoint of safety. During this period when adequate antiseptic treatment of the deep tracts seems precluded the discharge of pus into dressings not in contact with the wound and the attempt to minimize the infectivity of the issuing exudate by dichloramine-T strips within the drainage tube seems the most rational and safe procedure. An antiseptic necessarily must have actual contact with infected tissue in adequate quantity, and this may be accomplished 48 to 60 hours after operation by removal of one or both tubes, the fluid being introduced by catheter, placed to the depth of the tract. Just what Dakin's fluid introduced in 10 cubic centimeter amounts into these tracts accomplishes may be debated from many angles. A thorough mechanical flushing of the tract with fluid during the period of active infection may be credited with at least removing the detachable products of infection. We know that the infected fibrin lining the walls of the dead space eventually comes away in fragments or extensive sloughs, leaving the wall lined by clean granulation tissue. It has been claimed that Dakin's fluid causes a rapid disintegration of necrotic tissue. Our experience has been unqualifiedly in favor of this assertion, certain of the cases with definite necrotic abscess wall at operation healing without the discharge of macroscopic slough. Whether Dakin's fluid is actively germicidal, whether by its slight irritant nature more phagocytes are attracted to the infected locality, are questions of interest but rather indeterminable in a clinical study. Whether the action of the antiseptic in killing bacteria outweighs its destructive action on phagocytes, or whether the favorable outcome of wounds treated by Dakin's fluid is brought about despite the antiseptic rather than by reason of any inherent virtue are academic questions. Some investigators give the credit to the bactericidal influence of

the antiseptic; others to the phagocytic activity which goes on during the intervals between the injections. Whatever may be the action of the Dakin fluid, our results seem to indicate that, with continued flushing with 5 to 10 cubic centimeter amounts at 2 hourly intervals, these tracts cease to discharge pus and are lined by a clean granulating wall at an earlier period in the average case than when treated otherwise. When the period of suppuration has passed, the biologic-dynamic considerations of a clean dead space seem to favor immediate cessation of all treatment in order that wound healing may be most favored. Partial secondary suture (suture of the angles of the wounds) may aid in hastening closure; complete secondary closure will probably be disappointing.

There is one consideration which may be of greater importance than has been generally realized, namely the influence of the infection curve in the less resistant tissues of the abdominal wall upon the curve of infection in the tract lined by highly resistant peritoneum. It is conceivable that the rather large group of cases that surgeons drain when in doubt have their course greatly protracted by retrograde infection from the superficial portion of the wound. All dressing procedures, loosening, or removal and replacement of drains favor this contamination of the deep tract. Thorough antisepsis of the superficial wound with care in replacement of drains within the lumen of existing drains is well worthy of attention, whether or not one wishes to accept the responsibility of introducing an antiseptic into the intra-abdominal drainage tract.

In conclusion emphasis should again be laid upon the necessity of thoroughly mastering the details as well as the principles underlying the introduction of such antiseptics as Dakin's fluid into deep, infected abdominal drainage tracts. While a single accidental admission of 5 to 10 cubic centimeters of Dakin's fluid through an infected tract into the free peritoneal cavity would probably not result fatally, it would certainly be productive of intense pain and alarming symptoms. We believe that the probability of such a complication is slight if the treatment is carried out along some such precautionary lines as have been found safe in our cases. If one is not willing to give the time necessary to gain a correct theoretical understanding, and is not willing to give the cases his careful, personal attention at daily dressing, the method should not be employed as it will probably not only prove disappointing, but may result in serious complications.

DEPARTMENT OF TECHNIQUE

THE PROTECTION OF THE SKIN FROM PUS, URINE, FÆCES, CHEMICALS OR ANY OTHER IRRITATING MATERIAL BY THE USE OF SHEET RUBBER ADHERENT TO THE SKIN

By ANGELO L. SORESI, M.D., New York City

THE fact that the skin needs protection against prolonged contact with pus, urine, fæces, chemicals and any other irritating material, is as well known as the fact that an efficient protection is very difficult to obtain and that some patients have suffered more on account of the irritation of the skin following a suprapubic prostatectomy, or a pleurotomy, or the use of chemicals intended to sterilize some wounds, than on account of the original disease itself. We have been very successful in protecting the skin by adopting the following plan. Before the operation the intended line of incision is marked out, the skin is cleaned and dried with ether and painted with a solution of rubber cement dissolved in about five parts of ether, for about 2 centimeters all around the intended line of incision. To the skin so prepared is applied a piece of sheet rubber of sufficient size to cover the area of skin that might later come in contact with the irritating material. The sheet rubber is gently compressed against the skin, as it is done when a patch is put on a rubber glove, on a tire or any other rubber material. The surgeon then makes the incision in the usual manner, obviously first cutting the sheet rubber, then the skin and then the deeper structures. Along the line of incision the sheet rubber will have adhered to the cut edges of the skin, so that no material that could come out of the wound would

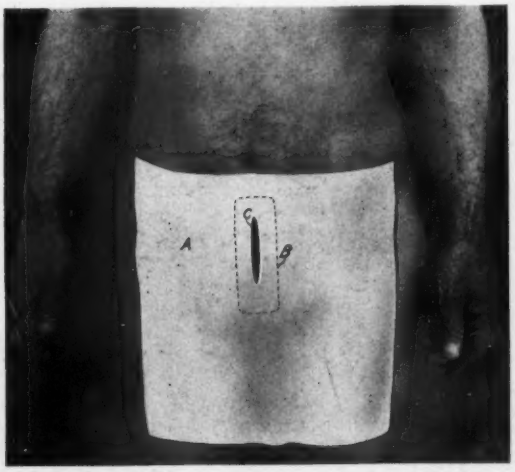


Fig. 1. Sheet rubber applied previous to the opening of the urinary bladder. *A*, Sheet rubber applied over the area of the skin that might become wet and irritated by the urine. Note that the sheet rubber covers completely the genitals. *B*, Dotted line shows how far from the intended line of incision the skin should be painted with the ether rubber solution. *C*, Incision through the sheet rubber.

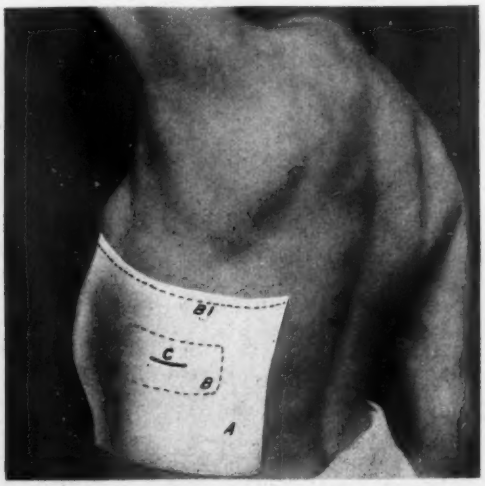


Fig. 2. Sheet rubber applied in cases of empyema. *A*, Sheet rubber. *B*, Dotted lines show the extent to which the sheet rubber should adhere to the skin, which has previously been painted with rubber ether solution. *B*^{1}, Shows adhesion of the sheet rubber to the skin above, so that the same should not fall if the patient should stand up. *C*, Incision through the sheet rubber.

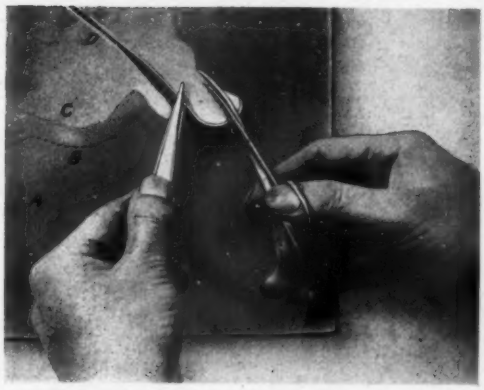


Fig. 3. Sheet rubber applied in cases of large wounds. *A*, Sheet rubber. *B*, Dotted line showing attachment of the sheet rubber to the skin. *C*, Contour of the wound. *D*, Line showing the sheet rubber cut in the middle, so as to allow its trimming along the contour of the wound with the help of thumb forceps that raise up the edge of the sheet rubber and scissors cutting very close to contour of the wound, as shown in Figure 4.

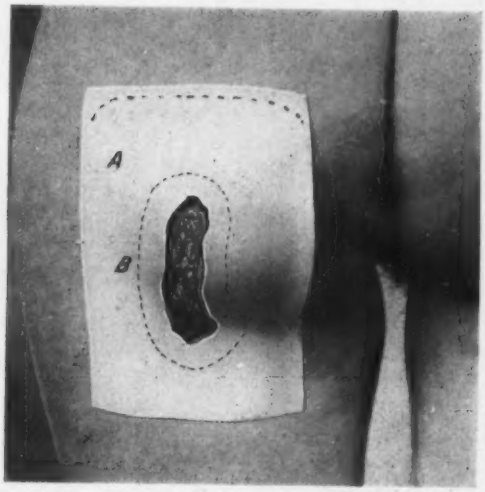


Fig. 4. *A*, Sheet rubber. *B*, Dotted lines showing attachment of sheet rubber to the skin. *C*, Wound with sheet rubber well trimmed around its contour.

ever touch the skin, which in this manner is completely protected from the contact with any irritating material. Illustrations show how sheet rubber is applied in different cases.

Precautions in the application of the sheet rubber. We have stated that the line of incision can be marked before applying the sheet rubber; this step, however, can be omitted, if the sheet rubber is rather thin, because through it all the structures and landmarks can be seen and felt very easily.

The application of the sheet rubber must be made so as to have the rubber adhere well to the skin. For the ones accustomed to patch rubber goods any advice is unnecessary, because the film of rubber that is left on the skin after the application of the rubber ether solution, makes the adhesion of any rubber material to it subject to the same rules that govern the adhesion of any patch over rubber material. In any event the precautions to be taken are: the skin must be dry before the application of the rubber ether cement, which must be perfectly dry and clean; the sheet rubber must be compressed against the skin so as to make it adhere completely without any air under it; if the ether cement solution is very thin, it is better to apply two or three coats of the same before applying the sheet rubber.

If the solution of ether and rubber has been allowed to stand some time and the sheet rubber is sterilized, the surgeon can cut through the sheet rubber without any previous preparation of the skin with tincture of iodine, because the wound will positively not be infected by the incision, after the skin has been protected with rubber ether cement. As explained in another paper,[1] we strongly deprecate the use of tincture of iodine previous to the painting of the skin with the ether rubber solution and the application of the sheet rubber which, we repeat, are sufficient to protect against infection.

When the surgeon has to deal with an open wound, such as the one shown in Figures 3 and 4 the procedure is the following: the skin around the wound is cleaned and then dried with ether; rubber ether cement solution is applied for about 2 centimeters around the wound; sheet rubber is applied over the whole wound, which can be filled, if deemed necessary, with sterile dry gauze. The sheet rubber is compressed against the skin; the sheet rubber is cut in the middle and trimmed all around the wound.

The sheet rubber must be of good quality; dentist dam is the best; common rubber tissue is too brittle.

If the skin needs only to be protected for a few days, the rubber cement ether solution applied rather thick, will be sufficient.

We advise having the sheet rubber adhere to the edge of the wound only for about 2 centimeters all around it, because obviously there would be no advantage in having all the sheet rubber adhere to the skin.

[1] Soresi, A. L., The preparation of the skin for operation with solution of rubber and ether instead of tincture of iodine. Ann. of Surg., 1910, Jan., p. 109.

AMPUTATION OF THE HIP-JOINT WITH REMOVAL OF THE WHOLE BONE, AND FLAPS AMPUTATED JUST ABOVE THE KNEE

By GASTON TORRANCE, M.D., Birmingham, Alabama

ABOUT two years ago a man, 30 years of age with a long standing tubercular condition of the whole thigh bone and also of the knee-joint, was referred to me for amputation at the hip. He had had the disease in some part of his leg since childhood and was very much emaciated and was quite toxic. An X-ray was made of the joint and showed destruction of the head of the femur with an ankylosis of the hip by means of an outgrowth of bone across from the trochanter to the ischium which flexed the thigh on the pelvis and drew it inward. He was a very poor subject for any kind of an operation and especially for a hip-joint operation.

Under ether anæsthesia I made an incision over the trochanter and down the thigh almost to the knee and removed the head of the bone and chiseled loose the bony connection with the pelvis and curetted out the acetabular cavity. The shaft of the bone was so much diseased that as soon as the head was gotten out, it broke in two just below the middle of the shaft and was removed. I then dissected out the femoral artery and ligated it with heavy catgut, without at any time during the operation using a tourniquet or any other means of controlling hæmorrhage. Up to this time the patient had not shown any symptoms of shock. With a large knife I now severed the fleshy part of the thigh just above the knee, and closed the flaps just as though it were an amputation at the knee-joint.

There was very little shock and the patient made a rapid recovery. He is now in perfect health and has a good firm stump (muscular). The photograph was made about a year after operation and shows him to be in better health than he has been in years.

I see the patient constantly and he is well and seems much pleased that he has a stump left.

EXTERNAL SURGERY OF THE NASAL ACCESSORY SINUSES[1]

By CORNELIUS G. COAKLEY, M.D., F.A.C.S., New York, and W. W. PEARSON, M.D., F.A.C.S., Des Moines, Iowa

IN external surgery of the nasal accessory sinuses, especially of the fronto-ethmoid group, we are confronted with two problems:

1. The division of the cases into those which are best approached by the external route, rather than by the intranasal.

2. The selection of operation which gives the most satisfactory end-results.

Let us first consider what cases demand external operative procedures. A discussion of this subject among those who have had a large clinical experience in the treatment of acute and chronic infections of the fronto-ethmoid sinuses, would probably result in a wide difference of opinion as to the value of intranasal or external operative procedures in any given class of cases. My own experience during the last 10 years is that it is possible to relieve or cure many more cases of fronto-ethmoidal disease by intranasal operation than I did in the preceding decade. The number of my external operations on the fronto-ethmoidal sinuses has diminished greatly during the past 10 years. Unquestionably bolder and more thorough intranasal removal of the anterior group of ethmoid cells, thereby establishing better drainage through the naso-frontal canal, and the teaching of a patient to pass a cannula into the frontal sinus for the purpose of irrigation, has cured many patients who I formerly thought could only be cured as a result of external operation.

As evidence of this during the past 11 years we have diagnosed 273 cases of frontal sinus disease, diagnosis having been confirmed in each case by roentgenograms. Of this number only 7 were required to have external operations. Of the total number of fronto-ethmoid cases operated upon by me 11 were patients who had been operated upon by surgeons and presented themselves after one or more operations with fistula, as evidence of the failure of the operation to eradicate the disease and secure the desired relief. Eliminating, therefore, these secondary operations, the percentage of cases requiring external operations is seen to be extremely small, viz., 13.5 per cent. It must not be inferred that the intranasal operation invariably cures the patient, if by cure one means complete cessation of all discharge, and the absence of more or less periodical recurrence of an acute process in the sinuses. So long as a patient has a frontal sinus that has been the seat of a chronic infection, it is possible for reinfection of that sinus to take place. If, however, the patient can be taught to irrigate his own sinus, or by a few treatments on the part of a rhinologist, can be relieved of his pain or discomfort, and brought back to a practically non-secreting condition of the mucous membrane lining the cavity, that patient will, in all probability, be satisfied with the result, rather than undergo more or less deformity as a result of an external operation. Orbital cellulitis, spontaneous fistula from the rupture of the anterior or inferior wall of the frontal sinus, presence of new-growths within the sinuses, patients whose symptoms persist in spite of attempts to establish satisfactory intranasal drainage, and those who cannot give the time to or refuse intranasal operations, are the only cases which, in our opinion, require external operation.

The most logical type of external operation on the frontal sinuses is that which was recommended by Killian some 17 or 18 years ago. Since it was first advocated various modifications of it have been suggested with a view of minimizing the deformity. The object of the Killian operation is the ablation of the entire infected mucosa and the obliteration of the cavities, thereby preventing future infections and recurrences.

The failures resulting from the performance of this operation that have come under my notice have been invariably due to the fact that the operator has left some portion of the mucous membrane in some but partly exposed part of the sinus. We have met with two types of imperfect operation: One is that in which the overhang has been left in the upper portion of the anterior wall, the operator being satisfied that he was able to remove thoroughly all the membrane underneath the overhang. If one examines many frontal sinuses he will easily see that this is a most dangerous procedure, because occasionally a little portion of the frontal sinus will extend upward through a small inlet and then expand out into a fairly good sized cell. This offshoot of the sinus is almost certain to be overlooked if any overhang is left. The other and more frequent source of failure has been the presence of a cell situated beneath the horizontal portion of the frontal

[1]Presented before the Clinical Congress of American College of Surgeons, New York, October 20-24, 1919.

bone, at the external portion near the external angular process of the frontal. This cell, or rather a prolongation from the ethmoid cells, may be entirely unsuspected unless it is most carefully looked for after widely opening and cleaning out the frontal sinus. A probe bent at right angles and passed externally from the naso-frontal canal parallel to the supra-orbital arch one-half inch below the arch, will usually find this cell. The anterior wall of this cell is usually flush with the posterior wall of the frontal sinus. The cell extends laterally beyond the outer margin of the vertical portion of the frontal sinuses. This cell is often overlooked when exenterating the ethmoidal labyrinth because it extends so far externally over the roof of the orbit. It often has a septum which runs anteroposteriorly over the roof of the orbit, and has but a small communication with that portion of the ethmoid labyrinth that is on the inner side of a line drawn to the middle of the orbit. Of 11 secondary operations performed during the last 11 years, every one of them had this large cell underneath the vertical portion of the frontal sinus. Roentgenograms of the frontal sinus do not always indicate the presence of this cell owing to the narrowness and to the dense frontal bone overlying it. The mucous membrane lining this sinus oftentimes fails to manifest much trouble for several years, until the engrafting of a fresh infection causes an increase in the secretion. Dense cicatricial tissue prevents the secretion from following the line of least resistance into the nose, and the swelling of the scar or upper lid, the evacuation of pus externally, is the eventual common history.

I would recommend for consideration a slight modification of the Killian operation that we have been doing since 1907, namely, separating the incision for the exposure of the vertical portion of the frontal sinus, and that for the ethmoid labyrinth. The former incision begins at the median line and extends in a slightly curved direction parallel with and just above the upper margin of the eyebrow, out to the very outermost portion of the external angular process of the frontal. The incision is carried directly down to the bone, the periosteum elevated above as far as the roentgenogram indicates the height of the frontal sinus, and the periosteum peeled downward one-eighth of an inch. This incision enables one to remove the entire anterior wall of the frontal sinus and to bevel the upper margin, so that there is no abrupt or rough margin for the periosteum to fall over on.

The incision for the ethmoid labyrinth is started one-quarter inch external to the supra-orbital notch, and about three-eighths of an inch posteriorly to the junction of the vertical and horizontal portions of the frontal bone. It is carried in a slightly curved direction downward, midway between the inner canthus and dorsum of the nose, down along the side of the nose to the inferior border of the nasal bone. From this incision the periosteum is reflected anteriorly and posteriorly elevating the pulley of the supra-oblique along with the periosteum. Through this incision ample space is given for exenteration of the ethmoid labyrinth, including the sphenoid cavity, and particularly making it possible to follow up all the cells of the ethmoid which extend laterally over the roof of the orbit, and which at the outer angle frequently communicate with the vertical portion of the frontal sinus, as heretofore described. The double incision has an added advantage in that one may readily coapt the wound without that puckering which is so frequently obtained in the single Killian incision, and permits one to remove the dressing from the ethmoid incision on the third day, while keeping up pressure by the bandage over the skin, of the frontal sinus so that there is little likelihood of infection taking place by way of the nasal passages.

Of the 35 cases upon which I operated primarily, three had recurrences, one at the end of two years, and the second recurrence seven years later. One operated on in 1904 had a recurrence in 1918. One operated on in 1910 had a recurrence in 1919. Of 11 cases operated upon by other surgeons and by me secondarily, none of them thus far has had a recurrence. Of the total 46 patients operated upon, 3 died; one operated upon in 1914 left the hospital apparently cured, when 3 weeks later he suddenly developed intense headaches, nausea, and vomiting; was admitted to the hospital and seen about 2 p.m. when he was in coma. Arrangements were made to operate upon him about 5 p.m. but he suddenly died with all of the evidences of what we took to be a rupture of a frontal lobe abscess into the lateral ventricle, which findings were confirmed by autopsy. The patient operated upon in 1908 died last fall during the epidemic of influenza, of pneumococcus meningitis. Examination of his nose the day before he died failed to show any evidence of sinus disease. The third patient, operated upon in April, 1918, died during the summer of 1919, cause unknown.

DISCUSSION BY W. W. PEARSON

From the beginning of my work in this line my tendency has been toward conservation; my first thought has always been to relieve the patient with as little operative work and display of interference as my judgment would permit. For this reason I have made use of the intranasal route rather than opening up externally with the resulting scar.

The submucous resection of the septum has aided me very materially in relieving sinus troubles through the intranasal route.

There are, however, types of cases that demand immediate operation and in the hands of most of us, the external operation is the safer. I refer especially to cases of sinus infection in which the interior of the nose is so disturbed through accident, unusual developmental defects, or severity of infective process that immediate action to save the life of the patient is demanded. I recall, for example, the case of a student whom I saw late one night with a temperature ranging from 104 to 105°, and delirious. It was his first experience with a frontal sinus infection. Following a Killian operation which revealed a sinus completely filled with a serosanguinous fluid, the relief was immediate and healing took place within a very short time.

An unfortunate feature in connection with the intranasal operations is that the irregular nose which usually means impaired drainage, is the nose more often subject to the sinus infection; naturally this means greater difficulty in operating intranasally.

In cases of orbital abscess secondary to nasal sinus infection, I have always felt that thorough drainage is so imperative that the external operation has been my selection.

As a student I learned of Professor Langenbeck's unfortunate removal of an eye. He operated under the impression that he was dealing with a new-growth, only to find an abscess the draining of which would not have necessitated the removal of the eye. I am not so sure that, in exceptional cases, the removal of the eye is not necessary.

Last summer a young man came under my care who gave a clear history of frontal sinus infection covering a period of 3 or 4 months; an orbital abscess complicated the condition. Evidently he had been desperately ill as he had no recollection of several weeks of the period of illness. Being on a farm in a remote section of Canada, the attending physician removed his eye and drained the abscess in a manner that probably saved his life. When I saw him he continued to carry a temperature and the orbital structures were in such shape that it was necessary to clean out the frontal and ethmoidal cells and do a little plastic work on the lids to permit the wearing of an artificial eye. The loss of the eye in this case was deplorable, but if I have the situation pictured correctly, this man owes his life to the practitioner who was not a specialist but who did recognize the importance of drainage even at the expense of an eye.

In discussing orbital abscess which, when present, has for its origin so often an infection of one or more of the nasal sinuses, I feel that the discussion would be incomplete if I did not mention the possibility of having to deal with blood cysts of the orbit, as pointed out by Doctor Gifford, or a chloromatous mass as was a recent experience with me; the latter case I saw last June.

A boy of about 12 years was referred to me because of a marked proptosis on one side. An examination of the nose revealed an organ regular in its makeup and free from any suggestion of sinus trouble; the X-ray indicated no sinus involvement; the blood examination, fortunately, showed a chloromatous change. This induced me to refrain from operation and later watch the development of the chloromatous condition with the invariable result — death.

The healing of a chronic nasal sinus empyema differs greatly from that of the acute condition, although I must admit that my judgment has often gone wrong in the prognosis of the latter. For example, of two cases which, according to their history had been active several months, the one healed with drainage within two weeks, whereas the second, while causing no apparent trouble other than that resulting from the drainage, still offers no promise of recovery.

Shall we operate upon all the chronic cases which refuse to heal under the usual careful attention? To define more clearly the latter group, I mean those giving rise to no constitutional symptoms but presenting more or less always the proof of their presence by the drainage in the nose. This question, I believe, can only be answered after explaining the condition to the patient and securing his decision. The ideal would be to restore every tissue to normal and do away with all infection. This sometimes entails extensive operations which are no doubt necessary in individual cases but should be avoided when possible.

The chronic cases giving rise to pressure symptoms or evidence of absorption must be operated upon when a sinus is large and filled with granulation tissue and pus. I recall a frontal sinus case operated upon by me several years since, the sinus measuring a little more than 12 centimeters, extending from the left external orbit across the forehead — I do not recall the exact depth, but it was exceptional; it was filled with the results of chronic inflammation — this

represents one extreme involvement. The sinuses not filled up with the results of chronic inflammation but secreting a thin mucopurulent fluid draining into the nose, represent the other extreme. The former demands radical operation; the latter occupies a place in the debatable field.

Several years since in conversation with a friend who has had an extensive experience in sinus work, he remarked that he had been impressed by the fact that the dura covering the anterior lobes did not seem to have the resistance to infection and to permit of the handling as does that in the temporal region. It has occurred to me that possibly the anatomical prolongations from the dura into the different foramina in this region, including those of the cribriform plate, have something to do, when under pressure, with increasing the susceptibility to infection and lowering the resistance.

No doubt the germ active in the individual case and the formation of the cell has much to do with the development of chronicity. Constitutional disease, syphilis, tuberculosis, etc., must ever be considered in our dealing with these cases and one very able assistant, the X-ray, should be used before any sinus operation is decided upon.

BRAIN ABSCESS COMPLICATING A LOCAL CRANIAL INFECTION

By WILLIAM SHARPE, M.D., New York City
Professor of Neurologic Surgery, New York Polyclinic Medical School and Hospital

IN discussing the condition of brain abscess, it must be remembered that a true brain abscess is a cortical and subcortical purulent formation, usually with but in the early stages without a definite limiting membrane or capsule; collections of subdural pus of a subdural abscess or of a localized purulent meningitis must not be confused with the condition of true brain abscess. The value of mortality and operative statistics of patients having the condition of so-called "brain abscess" has been greatly lessened owing to the greater frequency of subdural but extracerebral abscess formations and to their comparatively lessened danger to the patients, in that these subdural but supracortical abscess formations are usually well walled-off from the surrounding sterile subdural and subarachnoid spaces, so that they can be easily and safely drained directly through the dura; but to assert that a true brain abscess is present, merely because pus escapes upon puncturing or opening the dura of the mastoid area, frontal or sphenoidal sinus or infected fracture of the skull, is not warranted. It is only when the dura is adherent to the underlying cerebral cortex and the abscess cavity is within or beneath the cortex that the condition can be termed a brain abscess. It is this confusion of nomenclature which has rendered the statistics of the mortality of brain abscess of little or no value, since the mortality of true brain abscess is exceedingly high, whereas the mortality of localized subdural and meningeal abscess formations is less than 50 per cent.

The treatment of brain abscess is a surgical one, but its diagnosis and accurate localization are usually so difficult that any operation of drainage must be considered as an exploratory procedure; not only is this true of multiple metastatic abscesses in the brain as a complication of purulent foci elsewhere in the body, but also those abscesses of the underlying brain due to the extension of infection by contiguity of a fracture of the skull, gunshot injury, sinusitis and otitic disease. The great frequency of multiple abscess formations resulting from metastatic processes makes a most confusing clinical picture and the prognosis is always bad, whereas a solitary abscess of varying size is the complication of a local cranial infection. Fortunately in these latter patients, the subcortical abscess formation is frequently in close proximity to the primary infective process whether it be an overlying fracture of the vault of the skull, an underlying sinusitis of the frontal, ethmoidal or sphenoidal bones, or an ipsolateral otitic infection.

Unlike most brain tumors (excluding the gliomata) which produce an increase of the intracranial pressure by their added tissue formation or by a blockage of the ventricles, brain abscesses on the contrary replace brain tissue so that unless the escape of cerebrospinal fluids from the ventricles is blocked by a large subtentorial abscess formation, there are produced no signs of a marked increase of the intracranial pressure and this absence of high intracranial pressure is only of negative value in the diagnosis; a meningeal irritation, however, resulting from

the proximity of the abscess formation to the cortex will frequently cause an increase of the intracranial pressure and this serious complication of a possible acute purulent meningitis must always be feared; frequent cytological examinations of the cerebrospinal fluid are most helpful.

In the surgical treatment of brain abscess complicating a fracture of the skull, sinusitis, or otitic disease, it is essential to eliminate the original infective focus, and then (and this is the most important point to decide in the treatment of brain abscess of whatever cranial origin), if we are absolutely certain that the abscess formation lies directly beneath the affected dura and that this area of the dura is adherent to the underlying cerebral cortex, the ideal method of operative drainage is naturally through the site of original infection whether it be a fracture of the skull, a sinusitis of frontal or ethmoidal bones, or an otitis media with mastoiditis; that is, first we do the local operation to remove the original infective process from the area of the fracture of the vault, the frontal or ethmoidal sinuses and, in otitic disease which is the usual primary infective focus, the "cleaning out" of the mastoid cells and then exposure of the dura. The question now is whether this exposed dura should be punctured by an exploratory needle through the infected area of the fracture of the vault, the sinus or the mastoid cells in the hope that the brain abscess lies in the cerebral cortex just beyond the dura. I use the phrase "in the hope that the brain abscess lies just beyond the dura" advisedly, because in a large percentage of patients it is impossible to state with certainty that a cerebral abscess is present, and if it is present, to give its accurate localization. It is for these two reasons that the diagnosis of brain abscess, with few exceptions, must be a tentative one, and the operation of drainage must in reality be an exploratory procedure owing to the great difficulty of accurate localization even when the abscess formation is present. (The extradural abscesses as well as the subdural but supracortical and therefore extracerebral abscess formation must not be confused with the true brain abscess —a cortical and usually, a subcortical formation.) I repeat, if it is definitely known that the brain abscess lies in the cerebral tissue directly beneath the infected dura in which case the dura is frequently adherent to the underlying cortex, then the ideal method of drainage would be directly through this adherent dura into the abscess cavity itself—a cerebrodural route well walled-off by the adhesions of an earlier localized meningitis; but to attempt exploratory punctures through the dura, the sterile subdural spaces and into the cerebral tissue itself, in the hope of locating an abscess of the adjacent areas of the brain, and through the "dirty" infected field of the mastoid, or the infected sinus or the infected fracture of the vault, I say this method of operative procedure is not only lacking in surgical principles but, if the brain abscess is not found, it most assuredly aids in the formation of multiple subcortical abscesses as a result of the exploratory punctures and the extension of the infective process to the meninges so that an acute meningitis and meningo-encephalitis results. For fear of being misunderstood, it is only in those cases of brain abscess formation which lie directly beneath the dura of the infected area, such as the mastoid, and where the dura and the underlying cortex are adherent and well walled-off by the adhesions of a former localized meningitis that this method of opening the dura or puncturing it through the infected field of the mastoid should be advocated; if at the local operation of mastoidectomy or at the removal of infected cells of the sinuses or of infected bone of the fracture of the vault, there are found definite evidences of a subdural abscess in the color of the dura or of a cortical or subcortical abscess in the bulging, non-pulsating dura adherent to the underlying cerebral cortex, or if that most rare "stalk" of the abscess should be located, then in these patients and in these patients alone is it a rational and safe procedure to open or puncture the dura through this infected extradural field in the knowledge that the brain abscess lies directly beneath the operative area and that the sterile subdural spaces are well walled-off from the site of operation and drainage.

In a large percentage of patients, however, the local operation does not disclose any definite signs of a subdural lesion and the dura is not adherent to the underlying cerebral cortex, and these are the patients in whom it is distinctly dangerous to open or to puncture the dura and to explore intracerebrally in the hope that the abscess can be thus located and successfully drained. If the abscess is found by the first puncture opening, then its drainage through the sterile subdural spaces by a small opening is associated with the great risk of a purulent meningitis and if the abscess formation is not present or at least not located, then the danger of multiple abscess formations and a diffuse meningitis and meningo-encephalitis resulting from the exploratory punctures themselves is more than a probability; also, to puncture the dura one inch

or more above the infected field of the mastoid but through the same incision as the mastoid opening—this is *not* through a "clean" sterile area and it usually becomes infected from the mastoid as would naturally be expected.

It is in these fairly frequent cases that it is impossible to state with accuracy at the time of the mastoidectomy that the brain abscess lies directly beneath and contiguous with the dura of the infected mastoid area, and if the presence of a subtentorial and cerebellar abscess has been excluded (and cerebellar abscesses are the more easily diagnosed than the supratentorial and especially the temporosphenoidal ones), the operation of exploration of the temporosphenoidal lobe and the adjacent areas of the brain should be made through the "clean" subtemporal route as in the operation of subtemporal decompression and drainage. Naturally, the vertical incision should be used. If the brain abscess is not found, then the exploration has been performed with little or no danger of a resulting meningitis and at least a decompression has been afforded the patient in that the abscess may localize itself later. If the abscess is found, then it can be freely drained through the wide subtemporal opening with less risk of a meningitis occurring owing to the decompressive effect of the operation itself. If the abscess is in that part of the temporosphenoidal lobe adjacent to the infected mastoid area, then additional drainage of the abscess cavity may be obtained through this area. It has been my experience in these patients that cerebral tissues as well as the meninges are definitely resistant to infection from the drainage pus of the abscess itself if the intracranial pressure and the local pressure of œdema both from the abscess and from the operation itself, are not high, so that this exploratory operation of drainage through the subtemporal area is not only an efficient means of drainage if the abscess is found, but the complications of meningitis and meningo-encephalitis are greatly lessened. Besides this method of approach makes possible a much more extensive exploration so that if the abscess is not situated in the adjacent temporosphenoidal lobe, it can be satisfactorily drained if found in the ipsolateral frontal, parietal, or even occipital lobe. In my series of brain abscess cases, there are several of them which undoubtedly would not have been found if the exploratory puncture needle had been used through the mastoid area alone. As an efficient means of drainage, the double glass tubes, one tube within the other, so that the outer tube always remains in place in the abscess cavity while the inner tube can be removed and used as a means of suction-drainage, has been proved of distinct value.

In abscess formations of the cerebellum complicating otitic disease, the diagnosis and localization are usually not so difficult as in supratentorial lesions, and if at the operation of mastoidectomy with the dura exposed there are definite signs of an underlying cerebellar abscess either in the cerebellopontine angle or in the contiguous cerebellar lobe, then the dural drainage-puncture should be made directly into the abscess formation. However, if it is not definitely known that the abscess is situated subtentorially, then I believe no dural puncture should be made through the infected mastoid area but rather through a "clean" incision through the adjacent occipital area, that is, a unilateral suboccipital exploration and if necessary a bilateral operation.

CONCLUSIONS

The mortality of patients having the condition of true brain abscess is high; without operation practically 100 per cent, and with operation 70 per cent and even higher. Subdural and localized abcesses are excluded. The diagnosis of the intracranial condition and then the accurate localization are most difficult, and for these reasons the operation of drainage must always be considered as an exploratory procedure.

The ideal operative approach is the direct one—through the infected mastoid area, dura, and into the adjacent abscess cavity—but only in the presence of an adherent dura to the underlying cerebral cortex thus walling-off the infective process. In those other selected patients in whom the accurate localization of the abscess is not possible and in the absence of an adherent dura to the cerebral cortex, an exploration of the cerebral hemisphere should be performed through the "clean" subtemporal area, and, if the abscess is found, satisfactory drainage can be obtained. If the abscess is not located, then the risk of a resulting meningitis is practically nil, and it may be possible later to localize the abscess and to drain it. This operation, however, should only be used in these selected patients and the operation of mastoidectomy with wide exposure of the dura should always precede it in order to remove the primary infective focus and at the same time to ascertain the presence or not of definite signs of an adjacent brain abscess. If these signs are not present, however, the dura of this infected area should not be punctured in the hope of locating the abscess for the risk of a resulting meningitis is very high—whether the abscess is found or not.

TRANSACTIONS OF SOCIETIES

CHICAGO GYNECOLOGICAL SOCIETY

REGULAR MEETING HELD NOVEMBER 21, 1919, DR. A. H. CURTIS, PRESIDENT, PRESIDING

INVERSION OF THE UTERUS

DR. CAREY CULBERTSON: I desire to report, for the purpose of placing it on record, a case of inversion of the uterus. The patient was a primipara, 20 years of age and at term. I have not the date of her last menstruation, but she came into labor at 1 a.m. on October 4, 1916, with spontaneous rupture of the membranes. Delivery was accomplished at 7:15 a.m., October 6, 1916, by low forceps extraction, chloroform anæsthesia. The placenta could not be expressed spontaneously. Her attending physician waited 1 hour. Hæmorrhage then began and he introduced a pack. Hæmorrhage continued, the pack was removed, and the placenta manually delivered, after which the uterus was repacked. The records show that there was some continuous oozing until 12 noon on the same day. A second degree laceration of the perineum was not repaired.

In the afternoon of October 6, 1916, I was called to see this patient in consultation. She was pale; anæmic; pulse, thin and quick, 120 to 160; temperature normal. A mass was felt above the symphysis which was thought to be or was recognized at that time to be the uterine fundus. It felt hard and was well contracted through the abdominal wall. The vagina was tightly packed with gauze which was not stained by any present hæmorrhage. When I saw the patient she was in bed with the foot of the bed elevated. Strychnine had been administered, continuous normal salt solution was being given per rectum by the drip method and 3 pints had been introduced by hypodermoclysis. We regarded the case at this time as one of postpartum hæmorrhage in which the bleeding had been checked, and we did not disturb the packing which had just been put in.

On October 9, I was again called to see this patient. She had continued to bleed, the tampon had been removed, and the uterus again packed. At this time the abdomen was flat, soft, and relaxed with a concavity similar to a hernial opening palpable in the plane of the inlet. The patient was taken to the operating room and the gauze removed from the vagina. It was then discovered that the vagina was distended by a soft bulging mass which proved to be the inverted corpus uteri. The cervix could be felt as a tight ring high up laterally and posteriorly. An attempt was made to replace the uterus by taxis, but this was unsuccessful. The cervix was then grasped by two volsella and pulled down as far as possible, the inverted corpus being elevated behind the symphysis. The posterior cervical wall or lip was then split mesially up as far as the vaginal fornix. With the finger the vaginal wall was then dissected from the cervix and the cervix from the peritoneum. With this dissection carried out the free edges of the cervical incision were grasped and pulled down into the field of operation until the constricting ring appeared to be free. With the whole hand in the vagina, the fundus could not be replaced. The posterior incision was then extended until the cul-de-sac was opened after which the corpus was very rapidly replaced. The cervix was then brought down by volsella and the line of incision sutured from above downward. The cervix had not been lacerated. The posterior cul-de-sac was then closed and the edges of the perineal wound freshened and repaired. The patient was then given stimulants and salt solution. On October 11, I heard from the attending physician that the patient had died.

DISCUSSION

DR. PADDOCK: I would like to ask why the doctor did not remove the packing when he was called to see the case the first time. Perhaps I did not understand; perhaps he did remove it and repacked, but if he did not, is he sure that the packing was carried well up into the fundus and did not simply occupy the lower uterine segment? Also was ergot used?

DR. CULBERTSON (closing): In answer to Dr. Paddock, I will say that when I saw the patient the first time in the morning she was in bed, her pulse was not good—it was very rapid and thin. I was told that the packing had just been put into the uterus and the vagina was tightly packed. There was no evidence of blood showing through the vaginal pack. I did not think it wise to disturb the patient sufficiently to remove the packing; that is, to take her to the operating room and remove the packing. There was a mass felt above the symphysis which appeared to be the uterus. That was the reason I did not disturb her at that time. The evidence seemed to be that the patient was not bleeding at the time of my first visit. The case appeared to be one of postpartum hæmorrhage in which the hæmorrhage had been checked.

The question regarding the reposition of a uterus that is infected is always a proper one and that was taken into consideration. This uterus as it presented into the vagina was quite white and dry, but began oozing slightly as soon as replaced.

The cause of death was given as acute anæmia.

With regard to ergot, I have no notes here. As I recall, it was prescribed.

EPITHELIAL MASSES IN THE OVARY

DR. ARTHUR H. CURTIS: During the last two years I have been interested in a study of fallopian tubes. It often happens in the removal of tubes that one has to remove the ovary which is in close contact with the tube. In examination of more than one hundred specimens, I found in two cases, very small epithelial masses in the ovary, microscopic in size. These I think are unquestionably embryonal rests, which would later develop into dermoid cysts.

KIDNEY LESIONS IN GYNECOLOGICAL PRACTICE

DR. W. C. DANFORTH discussed the subject of kidney lesions in gynecological practice. (See p. 284.)

DISCUSSION

DR. T. J. WATKINS: I desire to call attention to the importance of the use of a method of *palpating the kidneys bimanually*, using much the same technique as is used in bimanual palpation of the ovaries and tubes. The usual method of causing the kidney to slip between the fingers is useful but is not sufficiently delicate, except in an occasional case. The other method permits one to obtain an intelligent idea of the size and sensitiveness of the kidneys in at least 95 per cent of women.

I also desire to emphasize the importance of instituting medical treatment for infection of the bladder and kidneys before resorting to instrumental examination, which always traumatizes more or less and which may result in contamination. A considerable percentage of cases of infection of the bladder and of the pelvis of the kidney will be cured with medical treatment. Should this prove inefficient, then the use of cystoscopy, ureteral catheterization, and possibly kidney lavage may be indicated. Our experience has been that the use of the above system has been very gratifying in a considerable number of cases.

DR. C. HENRY DAVIS: I have a patient who is of interest in connection with Dr. Danforth's paper. This patient was referred to me in the third month of pregnancy. At that time she gave a history of pain in the region of the right kidney of two years' duration. She had been told that she probably had a kidney stone. Examination of the urine showed albumin, a heavy trace, and microscopic examination showed both red and white corpuscles. The catheterized specimen of urine revealed a few colonies of colon bacilli and staphylococci. It was decided that she should be carried through with expectant treatment. She was delivered 18 days ago and for the first 9 days made an uneventful recovery. On the ninth day her temperature went up to 103°. For the next 4 days her temperature varied from 102 to 104.2°, with very severe pain in the region of the left kidney. An X-ray picture taken during pregnancy, showed a shadow about the size of a quarter in the region of the left kidney. Other pictures were taken shortly after delivery and showed the shadow of a stone in the same position. A catheterized specimen taken on the fourth day after delivery was sterile. A catheterized specimen taken after the flare-up in temperature was loaded with colon bacilli. It would seem in this case that the traumatism of labor so injured the kidney that there has been a recurrence of the colon bacillus infection in the traumatized kidney. The temperature has again returned to normal and we hope that she may subsequently come to the more simple operation for stone. One peculiar point in this case is that in the later weeks of pregnancy the patient was much more comfortable than she had been at any time within the past 2 years. She was always much more comfortable at night than during the day.

DR. BARRETT: I wish to speak of a patient I have in the hospital now, in whom infection and disturbance of the ureter took place as a result of a pus tube. The patient, a colored woman, entered the hospital in March, with multiple pelvic abscesses. The pelvis was what internes call a "frozen pelvis." We did not think the patient could stand a radical operation at that time. We drained the tube evacuating an enormous amount of pus. The patient was in the hospital until June and at no time did we consider her in a condition for a radical operation. We allowed her to go home and told her to come back if the infection recurred. She came back later, with a slight fever and enormous masses in the pelvis. Because of her improved condition, we felt justified in doing a radical operation.

Upon looking down into the pelvis it seemed as though the structures had all run together, and it was very difficult to distinguish one from the other. We worked down from the left side and found a pus tube in close attachment to the ureter, and removed the tube with difficulty. Elevating the uterus I came upon an enormous mass on the left side, which I worked out, taking the broad ligament with it. I put a forceps on the broad ligament on each side and rolled the tumor out and found that the ureter had been severed. The ureter had been pressed upon and infected by the mass and was friable and had been pushed up so that it lay close to the uterine artery. At first I thought we would leave the ureter, allowing it to drain and later probably take out the kidney. However, upon pressing the ureter down, while it was entirely free from the bladder up to the pelvic brim, we found the little narrow end and did an end-to-end-anastomosis. I then gathered enough peritoneum from different sources to make a covering for the ureter and put a drain-

age tube down deep enough so that we could secure proper drainage from the ureter. It leaked profusely for a few days and then leaked less and less. The patient is recovering, the ureter is intact, and there is no leakage of urine and no enlargement of the kidney.

DR. CURTIS: Is there urine coming out of that ureter?

DR. BARRETT: With the drainage tube in she was passing about 18 ounces of urine from the bladder. As the urine ceased to come through the drainage tube she passed more and more urine from the bladder and she is now passing about twice the amount. We have not cystoscoped her.

DR. JOSEPH L. BAER: I want to cite briefly two cases indicative of the way in which we are often misled.

Four months ago a patient was referred to me, who ever since her first pregnancy (she has now two half-grown children) had a mass in the region of her left kidney. Her physician had made innumerable analyses, all of them showing albumin, pus cells and occasional red cells, but she had always avoided having a more accurate diagnosis. Finally he impressed on her the importance of a more definite investigation. During this entire period she had one outstanding symptom: constant backache. The backache was so severe as to make her a semi-invalid although she was naturally a very vigorous and active woman. She went up to Rochester and the Mayos did a laparotomy and diagnosed the case as one of congenital cystic kidney. They closed the wound without further intervention. Some months afterward she saw me and because her backache was so typically sacral in character and as she had a distinct retroversion with the uterus, a relaxed perineum, and relaxed supports of the uterus, I felt disposed to consider the backache unrelated to the left kidney mass and suggested to her physician that I thought with plastic work her backache could be relieved. Under a general anæsthesia I suspended the anteflexed uterus to the abdominal wall and then did a perineorrhaphy. She is entirely relieved of her backache. Clinically she is well. She still has the so-called congenital cystic kidney with albumin, pus, and blood.

The other case I encountered some 5 weeks ago in a woman, aged 62, who was brought in with the clinical picture of acute renal colic. She had blood in the urine; she had intense pain, radiating from the high right lumbar region down the groin into the labium; she had a slight temperature and moderate leucocytosis. Her abdominal symptoms became more definite and 36 hours after the onset of the attack she was operated upon for acute appendicitis. A suppurative appendix was found overlying the ureter. She was a diabetic with a blood pressure of 220 and 10 days after operation she suddenly became very much worse and again developed the right-sided pain. Before operation a bimanual examination revealed an entirely free pelvis with only the stump of the cervix left following a hysterectomy some twelve years previously. The second bimanual examination after the onset of the pain revealed a beginning Douglas abscess, which was opened a week later. Her pulse ranged from 140 to 160. She had temperature and repeated chills. The Douglas abscess was evacuated. It is now 3 weeks since the second operation and in spite of the 200 blood pressure and the diabetes she is better, notwithstanding the fact that she developed a fæcal fistula which persisted for some 6 or 8 days and then apparently closed, there being now only a superficial abdominal opening.

DR. CURTIS: One or two points brought out by Dr. Danforth seem to me to merit further consideration. The first question is whether all these cases of pyelitis of pregnancy are the result of pressure upon the ureter. We very often have residual urine in the bladder with a considerable amount of pus. I wonder if they are not the result of residual urine.

About the use of disinfectants in washing out the ureter, I believe it has been the experience of urologists that it makes little difference what solution is used and that sterile water does as well as anything. Again, I think there has been a tendency recently to believe that irrigation of the kidney pelvis is not as helpful a measure as we formerly thought. In a recent conversation with Dr. L. E. Schmidt he told me he had given up pelvic lavage years ago.

DR. PADDOCK: The pressure of the pregnant uterus on the ureter causes infection. I have found in several cases of pyelitis in pregnancy that the knee-chest position several times a day was very beneficial and often curative.

DR. DANFORTH (closing): In the question brought up by Dr. Watkins as to the value of palliative treatment, I perhaps gave the impression of being more radical than I really am. I think in the acute stage when the patient comes into the hospital with high fever, she should be put to bed and treated. Many come in with pyuria without any temperature. In these we can immediately proceed to discover whence the pyuria arises. Dr. Davis spoke of a case where the temperature went up after delivery. I think it is very common to have a rise of temperature sometime during the first week. On the third, fourth or fifth day the temperature will shoot up and then come down, and this happens so frequently that you look upon it as a typical curve of a flare-up of a pyelitis. We do nothing for that except give urotropin. If the pyuria does not disappear after the patient is well and up and about, then we attempt to find out the cause.

I had an experience similar to Dr. Barrett's. I had a patient with a mass on the left side which was enucleated with difficulty. I also enucleated a part of the ureter and did an end-to-end anastomosis and the patient recovered, but had a leakage which did not disappear and finally necessitated removal of the kidney which finally we did and the woman is now alive and well. She had been under Dr. Babcock's care for a rapid heart. I saw another

case in which a surgeon took out a stone from the left ureter and a urinary fistula followed, which lasted for some time but healed up. This woman has a pyuria with colon bacilli. In a recent case in which I thought I did cut the ureter, I did a cystoscopy and found the ureteral opening discharging as usual. From the urologists I think we can get almost any opinion you like about irrigating the kidney depending on whom you ask. I recently had a talk with Dr. Kretschmer and he was as positive as one could be that any case of pyelitis or infection of the kidney which did not promptly heal should have lavage. He used a much stronger solution than I am in the habit of using.

The knee-chest position has been used for a long time and I have had many women go through without trouble. I think it is exceptional to have a woman abort.

As to the question of pressure upon the ureter, in spite of discussions in the literature to the contrary, I really think that pressure does exist, as evidenced by a case I reported. She was a doctor's wife, who had a marked pyelitis with pregnancy. In the attempt to carry her through her pregnancy we put a catheter in the right ureter up to the pelvic brim, but it would go no farther. After turning the woman on her left side the catheter passed up easily. This, I think, is definite proof that pressure existed upon the ureter.

As to the question of hæmaturia, I had not thought about it. I think the most logical conclusion would be that it would bear a relation to infections of the urinary tract.

MATERNAL MORTALITY

DR. C. H. DAVIS read a paper on "Maternal Mortality." (See p. 288.)

DISCUSSION

DR. C. S. BACON: The pessimism of the author is easily understood. There is a reason for it but I doubt whether it is quite justifiable. I used to feel the same way when I was young, some 20 years ago, when I made a study of conditions in Chicago in a paper presented to the society. But physicians were not properly educated years ago in the schools, and there are still many in general practice. I hardly know how they are to be reached except through the hospitalization of obstetrical cases, in which respect there is certainly a great improvement. There are a great many more hospitals in this city, for example, and there is an enormous increase in the number of obstetrical cases going to the hospital in the last 20 or 30 years. With the improvement in the standardization of the hospitals which is now going on, which is under the stimulus of the work of the medical associations, especially the American College of Surgeons, and under the stimulus of the rules governing obstetrical technique, obstetrical practice is found to improve greatly. If we can help to increase the number of cases confined in hospitals, we will be doing good. I have no doubt that there will be an increase in the future. The great difficulty is, of course, with the poor and those in rather moderate circumstances—the cost and the difficulty of getting mothers away from their families. Anything that can be done to increase the number of beds devoted to obstetrics in hospitals for moderate cost is very important.

DR. PADDOCK: I am convinced that these statistics are misleading. It seems to me looking back over the past 20 years that there has been improvement. The young men who have gone out into practice have certainly improved in asepsis, treatment, and skill and care in handling these cases. I cannot believe that the mortality is as great in the hands of the young men who are graduating today as it was 20 years ago. As the essayist has said, he saw only three cases and now they see 10 or 12 or 20. They handle some of these cases. I am not convinced that this mortality is entirely with the physician, but more with the midwives who have not improved in their treatment of cases. I would like to know the mortality percentage in the hands of midwives and in the hands of doctors. Also, I would like to know if these statistics are made up from abortions, that is, mortality resulting from abortion.

DR. C. H. DAVIS (closing): I am very sorry, Dr. Paddock, but the statistics are against you. If your statement is judged by statistical reports your teaching has been of no avail. Dr. Meigs answers you on page 17 of her report:

"During the 23 years ending in 1913 in this country no definite drop in the death rate caused by pregnancy and confinement can be demonstrated; nor can any decrease in the death rate from puerperal septicæmia be shown."

Relative to midwives, you will find in the report of Dr. Williams, which was made to the American Medical Association in 1911, and published in the *Journal*, January 6, 1912, Vol. 58, page 1, that he comes to the conclusion doctors are as much responsible for the continued high maternal mortality.

During the past 60 years the number of cases delivered by midwives has been constantly decreasing, but the general maternal mortality has not decreased. You may find tables in the *Transactions of the Edinburgh Obstetrical Society*, 1910–11, xxxvi, 26, which show that the maternal mortality has not decreased in the last 60 years. I reproduced these tables in my little book on *Painless Childbirth* published in 1916. There has been a great decrease in the number of deaths from puerperal sepsis in hospitals. In early days great waves of puerperal sepsis swept through the hospitals and once started practically every woman developed an infection. At that time in no maternity hospital was there a death rate of less than 10 per cent and from that up to 50 per cent. But during the past 30 years the maternal death rate in hospitals has been cut down, until at the present time only a fraction of one per

cent of the patients cared for, even including those brought in from the outside infected, die from puerperal sepsis. Yet, according to all available statistics, the number of women who are dying from childbirth and puerperal sepsis remains as high today as 60 years ago.

Any of you who have not read the bulletin on Maternal Mortality, published by the Children's Bureau of the Department of Labor, should get it.

The ratio of maternal deaths per 1,000 live births in 1910 was 6.5, that is 6.5 mothers died for every 1,000 live births in the registration area. In 1916 it was 6.6. The average mortality from puerperal sepsis for the years 1901–5 was 6.3 maternal deaths per 100,000 population. In 1913 it was 6.0 and in 1916, 6.7 per 100,000 men, women and children. Now, gentlemen, these figures are available. We are all inclined to believe that because we in our practice rarely have puerperal sepsis, because we do not lose many cases from this cause in our hospitals, that deaths from puerperal sepsis are decreasing. Statistics indicate they are not decreasing; they have long remained stationary. It is time that we who are interested in diseases of women make a concerted and continued effort toward bettering this condition.

INFECTION OF PELVIC ORGANS AFTER RADIOTHERAPY

DR. CURTIS: The uterus that was removed this morning had been treated a few days ago with 50 milligrams of radium for 24 hours. I opened the uterus this afternoon. There was a cancer of the cervix which had not extended to the fundus. The radium had so affected the endometrium of the fundus that it was changed into a gray-green foul smelling sclerotic mess. This illustrates that infection of the pelvic organs is not unlikely after radiotherapy.

DR. HAROLD O. JONES read a paper on the "Use of Radium in Pelvic Work."

CHICAGO SURGICAL SOCIETY

REGULAR MEETING, NOVEMBER 28, 1919, DR. CHARLES E. KAHLKE, PRESIDENT, PRESIDING

DR. EMIL G. BECK read a paper entitled, "A Further Report on the Treatment of Deep-Seated Recurrent Carcinoma by the Denudation Method."[1]

DR. SAMUEL C. PLUMMER reported three elbow cases, exhibited plates, and showed lantern slides of the same.

DR. EDWARD H. OCHSNER read a paper entitled, "A New Skin Suture Material."

DEVICE FOR MAINTAINING APPOSITION IN FRACTURES

DR. JOSEPH F. SMITH, Wausau, Wisconsin: I wish to show a little device for maintaining apposition in fractures of bones in cases where there has been extensive crushing injury and exposure of the bones, with loss of bone substance.

My associate (Dr. Jones) was connected with Base Hospital No. 1 in New York, where they handled a large number of compound fractures, and they attempted to use a combination of the Lane plate with the Parham-Martin band, but were not successful because of the tendency of the plate to slip laterally. This device consists of a plate made of vanadium steel, having some sharp pointed spurs which we think will aid in eliminating the difficulty of lateral displacement.

I will simply pass the plates around for inspection.

We use a Parham-Martin band through the slots in the steel. The spurs can be driven into the bone to prevent lateral slipping. When the Parham-Martin bands are tightened the plate is held firmly in place and the spurs serve to prevent the lateral slipping.

These plates are designed to be used only during the period of antiseptic treatment of the wound and may be easily removed as soon as the wound is clear and callus sufficient to hold the bones in apposition has been laid down.

INTRACRANIAL PRESSURE

DR. CASSIUS C. ROGERS read a paper on intracranial pressure. (See p. 291.)

DISCUSSION

DR. KARL MEYER: I agree with what the essayist has said in regard to intracranial pressure. A number of these cases have presented themselves at the Cook County Hospital from time to time.

About four months ago a man, 70 years of age, sustained an injury to the frontal lobe, causing a marked depressed skull fracture. He was sent to the Columbus Hospital where an attempt was made to trephine him and raise the depressed bone at least two inches in the frontal lobe. He was subsequently admitted to the Psychopathic Hospital with symptoms of paranoia. He was sent to the surgical service of the Cook County Hospital, and the depression raised. Shortly after operation he was dismissed from the County Hospital without any symptoms of paranoia and has remained well for six months.

DR. EDWARD H. OCHSNER: There are in the State of Illinois literally hundreds, yes thousands, of young people who are suffering from some defect which makes it difficult, if not impossible, for a large percentage to adhere to the ordinary standards required by society. If what Dr. Rogers has told

[1] Surg., Gynec. & Obst., 1919, xxix, 325.

us this evening is applicable in a large percentage of these mental defectives, it is one of the most remarkable things that has ever been discovered.

There are unquestionably many people who lie and steal because that is the way their ancestors made their living; they are simply schooled in that respect. There are many young people from good families whose ancestors have come through a lying and stealing period of many generations, who have short periods in their lives when they lie and steal and cannot help it. These young people are the most unfortunate and the most difficult ones in our society because they come in contact with the courts. They are sentenced to our juvenile institutions, which unfortunately are often schools for crime. Their experience makes them antisocial. They become anarchists; they are down on government; they are down on society. They are mighty bright and clever people, and they think that they are sent out into the world to "do" those who have done them. You cannot blame them very much. After one has been wronged two or three times, if he has any ginger in him, he is apt to hit back. These people have been injured by society; they have become so bitter they hit back.

The other class does not amount to much. They resort to petty thieving. They are not real, serious criminals. Most of you have had in your practice boys and girls who have done foolish things. Some of them come in contact with the law. If we could find a cause for producing such crimes and remove it, we would render an enormous service to society. I believe the solution of this problem is up to the medical profession. Criminologists have arrived nowhere. Crime is steadily on the increase throughout the world. The question of crime in Chicago and Illinois is a stupendous one, and if it can be solved, it would be the biggest problem that has been solved by the medical profession in the last twenty years.

Dr. Jacob Frank: I would like to say a word or two about traumatic intracranial pressure. Pressure may be due to an effusion extradural or intradural. It may involve the arachnoid or the brain tissue itself, or the lateral ventricle.

I have had occasion to open the skulls of a number of patients with slow pulse. A good deal depends upon where the fluid is situated. If it is extradural and you open the skull, you will get a free flow of fluid, and you will note immediately the effect of the slow pulse while the patient is on the table. You will observe the increase in pulse rate; the respiration will become better. If this does not take place, it is feasible to open the dura. I have seen cases where the fluid was between the dura and the arachnoid. If the patient does not react on the table, and the symptoms continue, it is advisable to tap the lateral ventricle. I have done that and have obtained fairly good results. I have punctured the spine without any beneficial results.

We know from the intracranial pressure experiments that have been made of injecting fluid into the lateral ventricle or subdural space, it will not pass into the spinal canal or vice versa. Many years ago, at St. Elizabeth's Hospital, when we had a great many cases of head injuries from railroad, horsecar, and cable car accidents, I demonstrated that point on the operating table. It is altogether different from intracranial pressure due to some pathologic condition. It does not do any harm to remove a part of the skull. If there is any hæmorrhage, it is quite a different matter. If we go back to the old classification of concussion of the brain, we know these patients will die, and if we read the old books on this subject we are told that no pathology in the brain is found. If you open the dura, you will see a small petechiæ. What good would it do to raise a part of the skull if pressure were in the ventricle? Those are the patients that die. Those patients in whom you simply raise the skull and see fluid escaping, and there is no internal pressure, get well. The extreme cases are those in which the pressure is in the brain itself and in the lateral ventricle.

Dr. Rogers (closing the discussion): I see a good many cases of fracture of the skull. I see cases that have been in the hospital as long as twenty-one days unconscious, with fracture of the skull, and nothing has been done except repeated tapping of the lumbar region. One man was tapped many times to relieve intracranial pressure, and died on the twenty-first day. I believe that man's life could have been saved by an early decompression, opening the skull and establishing extradural drainage.

With reference to the remarks made by Dr. Ochsner, I believe every one of these cases instead of being sent first to a reformatory should be sent to a pathologic laboratory for an examination, to see if anything can be done for them. I have never known of a man being reformed in a reform school.

CORRESPONDENCE

TEMPORARY STERILIZATION OF WOMEN

To the Editor: Under the above title in the December number of SURGERY, GYNECOLOGY AND OBSTETRICS, Dr. Turenne, of Uruguay, describes the technique of tubal sterilization as performed for the first time by him in October, 1916. In his article he questions the originality of this procedure, but says that so far as his knowledge goes, the operation has never been previously performed.

My first operation of this character was done on November 15, 1913, at the Polyclinic Hospital, the technique being identical with his, so far as I can judge from his description.

The history of my case is as follows: Mrs. L. J. W., age thirty-two, married eleven years, during which time she had given birth to three full-term children by instrumental deliveries, and had had two miscarriages at five weeks, cause unknown. She presented a lacerated and greatly relaxed vaginal outlet, with a well marked cystocele and rectocele. The cervix was lacerated, hypertrophied, and eroded. The uterus was small, anterior, and freely movable. At the operation the cervix was dilated and the uterus curetted. A bilateral trachelorrhaphy and perineorrhaphy was performed. The abdomen was then opened by a transverse suprapubic incision. The left ovary enlarged, cystic, and prolapsed, was resected. The right ovary and both tubes were normal.

The patient had requested sterilization and this was performed by embedding the fimbriated ends of each tube in a pocket on anterior face of broad ligament, holding in place by a continuous enclosing suture of fine silk. Convalescence uneventful.

Normal marital relations were resumed and continued until January, 1916, when she menstruated normally on January 17. The February and March periods were missed, and when I examined her on March 9, the uterus showed slight enlargement but no softening. There had been marked nausea and vomiting during the preceding month with beginning pains in the breasts. In the region of the left adnexa there was a globular tumor the size of an egg, that was markedly tender on palpation. She had been spotting every day during the previous month, a symptom which she had never had before. A diagnosis of ectopic pregnancy was made, and on March 11, the abdomen was opened through the scar of the old incision. A two-inch cyst of the left ovary was removed.

A transverse fundal incision was then made in the uterus, and what was, apparently an interrupted early pregnancy, with numerous old and new blood clots, removed. In this no fœtus was discovered, but the pathological report returned from the laboratory showed evidences of pregnancy.

The uterine incision was closed and the proximal ends of the tubes tied with linen thread sutures and cut, in order to make certain the sterilization attempted at the first operation three years before.

At this time I carefully examined the site of the embedding of both tubes. On the right side there was continuous union between the broad ligament and the circumference of the tube, and at no point could I discover any point of communication between the peritoneal cavity, and the embedded ostium of the tube. On the left side the embedding was equally perfect with the exception of one point anteriorly, where a minute opening, pin point in size, was seen. With a little force this opening admitted the passage of the smallest sized filiform bougie. Here was the means of communication between the abdominal cavity and the ostium of the tube, through which the ovum must have passed.

In using this technique for temporary sterilization, I was guided by the experience gained in attempting to relieve sterility where gonorrhœal infection had resulted in the so-called "clubbed tubes." In opening up these tubes at operation, I had repeatedly found that the fimbriæ could then be released, apparently as normal as ever. Some cases so treated have borne living children.

It therefore occurred to me, as it did to Dr. Turenne, that a good way to produce temporary sterilization, would be to close off the fimbriæ by embedding them between the folds of the broad ligament. I embedded them in my case on the anterior face of the broad ligament, so that, if by any chance the occlusion should not be complete, the opening would be at so great a distance from the ovary, that there would be little likelihood of an ovum entering.

But nature proved herself equal to the emergency. The ovum was carried to the anterior face of the broad ligament, presumably from the diseased ovary on the same side, and gained access to the embedded ostium of the tube through an opening so minute as to be almost imperceptible to the naked eye.

In view of further experience with these cases, I am inclined to think that a surer and better way of accomplishing temporary sterilization would be to invert the fimbriæ into the tube and close the lumen with a purse-string suture.

NEW YORK CITY CHARLES G. CHILD, JR.

AMERICAN COLLEGE OF SURGEONS

HOSPITAL STANDARDIZATION

HOSPITAL standardization under direction of the American College of Surgeons comes into its own. The minimum standard of the College, together with the progress now associated with it throughout the continent, makes up one of the great events in modern medicine. "The College," said Dr. Henry S. Pritchett, president of the Carnegie Foundation, in a recent interview in New York, "has advanced hospital conditions on this continent by ten years." In the following pages are given some excerpts from a recent report of the College (Bulletin Vol. IV, No. 4). This bulletin is of interest to every doctor.

The College has won by its disinterestedness. All of its work has gone for the better care of patients in hospitals. No specialty of medicine has benefited under its hospital program more than any other specialty. The breadth of view of the College and its idealism without sentimentality or "uplift" have won the support of doctors and of hospital workers. The work goes forward today with cumulative force.

With regard to the minimum standard, we quote from the bulletin: "It grew out of straight thinking of the clearest minds in medical and hospital work on this continent. It is practicable, workable, and constructive. It costs efforts rather than money. It safeguards the care of every patient admitted to the hospital by insistence upon competence on the part of the doctor, by thorough study and diagnosis in writing for each case, and by a checking up, at least once each month, of the clinical service of the hospital. It fixes responsibility throughout the hospital. It calls for the 'production sheets' of the hospital. It encourages and even compels research. It defines the minimum service to the patient which, beyond all debate, is considered essential."

The American College of Surgeons, at its beginning in 1913, included among its purposes the betterment of medical education and of the clinical practice of medicine. It recognized that matters of public health, the prevention of disease, and the intelligent distribution of the benefits of medicine are inseparable from these purposes. Work in these fields is known as hospital standardization. The following pages are a report of progress for the year 1919.

The administrators of the College took up hospital standardization with no preconceived idea or theory as to what should be done. They decided to study actual conditions, to find out what the normal processes of growth are and how these processes could be given speed. No "announcements" were made of this plan. But for two years a preliminary analysis was quietly pursued.

During these two years the College accumulated data from hospital folk and doctors; also the judgments of these groups as to what work could most wisely be undertaken, all of which served as a basis of procedure. These "assets" were gathered from every state in the Union and from every province in Canada. They grew out of conferences with hospital staffs, with city, county, and state medical societies, with superintendents, and with hospital trustees. In other words, the program of the College was built upon the cumulative wisdom of those concerned with it. As the climax to this preliminary work, the State Committees on Standards met in Chicago on October 19 and 20, 1917; and out of this conference grew the General Hospital Committee which met in Washington, December 8 and 9, 1917. Some details of these two meetings are given on page 11, Bulletin Vol. IV, No. 4.

After these two meetings the program of the College took more concrete form. That

program was, first, to define a minimum standard; second, to enlist the co-operation of the hospitals in the fulfillment of the standard, this work to be accomplished through personal visits to the hospitals by staff members of the College; and third, to publish from time to time the list of hospitals throughout the two countries which fulfilled the minimum standard. The publication of the list, however, was not to occur until the hospitals themselves generally approved of such publication, each hospital having been given full opportunity to meet the standard under normal conditions. The College anticipates at this time the publication in October, 1920, of a list of the general hospitals of 100 or more beds which fulfill the standard. A similar list of the smaller hospitals is to follow later.

The minimum standard, in accordance with this plan, was then defined. But this standard, as just explained, has been in process of coming gradually to light. It grew out of straight thinking of the clearest minds in medical and hospital work on this continent. It is practicable, workable, and constructive. It costs effort rather than money. It safeguards the care of every patient admitted to the hospital by insistence upon competence on the part of the doctor, by thorough study and diagnoses in writing for each case, and by a checking up, at least once each month, of the clinical service of the hospital. It fixes responsibility throughout the hospital. It calls for the "production sheets" of the hospital. It encourages and even compels research. It defines the minimum service to the patient which, beyond all debate, is considered essential.

Above all, the minimum standard is designed to bring a sense of responsibility to those who have to do with a hospital, that each patient admitted receives care scientifically sound. It is on this basis that the hospital may seek the confidence, good will, and support of its community; and it is through progress in this line that the medical profession may most swiftly advance to its rightful position in society.

The minimum standard as it has been adopted follows:

THE MINIMUM STANDARD

1. That physicians and surgeons privileged to practice in the hospital be organized as a definite group or staff. Such organization has nothing to do with the question as to whether the hospital is "open" or "closed"; nor need it affect the various existing types of staff organization. The word *staff* is here defined as the group of doctors who practice in the hospital inclusive of all groups such as the "regular staff," the "visiting staff", and the "associate staff."

2. That membership upon the staff be restricted to physicians and surgeons who are (a) competent in their respective fields, and (b) worthy in character and in matters of professional ethics; that in this latter connection the practice of the division of fees, under any guise whatever, be prohibited.

3. That the staff initiate and, with the approval of the governing board of the hospital, adopt rules, regulations, and policies governing the professional work of the hospital; that these rules, regulations, and policies specifically provide: (a) That staff meetings be held at least once each month. (In large hospitals the departments may choose to meet separately.) (b) That the staff review and analyze at regular intervals the clinical experience of the staff in the various departments of the hospital, such as medicine, surgery, and obstetrics; the clinical records of patients, free and pay, to be the basis for such review and analyses.

4. That accurate and complete case records be written for all patients and filed in an accessible manner in the hospital, a complete case record being one, except in an emergency, which includes the personal history; the physical examination with clinical, pathological, and X-ray findings when indicated; the working diagnosis, the treatment, medical and surgical; medical progress; condition on discharge, with final diagnosis; and, in case of death, autopsy findings when available.

5. That clinical laboratory facilities be available for the study, diagnosis, and treatment of patients, these facilities to include at least chemical, bacteriological, serological, histological, radiographic, and fluoroscopic service in charge of trained technicians.

AS MEASURED BY THE MINIMUM STANDARD

During the years 1918 and 1919 visitors from the College, in accordance with the foregoing program, visited the general hospitals of 100 or more beds in the United States and Canada. This list includes 671 institutions, the names and locations of which are given in Bulletin Vol. 4, No. 4.

The following table indicates in condensed form the findings of the survey in the hospitals of 100 or more beds. In this table 671 hospitals are considered. Of this group 198 hospitals meet today the minimum standard. Many of these hospitals have been visited twice during the last two years, and in such cases the second report was utilized in the table. In many instances, also, the hospitals pledged themselves to fulfill the minimum standard at the earliest practicable date and later made a signed report of the extent of their success. The reports of the hospitals received in this way are also embodied in the table.

STAFF MEETINGS, CASE RECORDS, AND CLINICAL LABORATORIES IN GENERAL HOSPITALS OF 100 OR MORE BEDS

1. IN UNITED STATES

Groups by Number of Beds	Hospitals in Each Group	Staff Meeting Each Month	CASE RECORDS							Clinical Laboratories		Hospitals Meeting Min. Stand.[1]
			Personal History	Phys. Exam.	Working Diag.	Lab. Findings	Treatment or Operation	Progress Note	Final Diag.	Path.	X-Ray	
100–149	282	91	131	117	112	181	178	112	194	170	174	66
150–199	133	49	67	56	54	95	93	56	97	96	95	37
200–249	65	27	30	31	27	40	40	27	40	40	40	22
250–299	57	33	32	31	28	42	41	28	44	44	42	24
300–349	24	13	13	12	12	18	18	14	20	21	20	8
350–399	11	6	6	6	5	6	6	5	6	6	6	3
400 or more	45	34	39	39	35	41	41	37	41	41	41	30
Totals	617	253	318	292	273	423	417	279	442	418	418	190

2. IN DOMINION OF CANADA

100–149	26	3	4	7	4	6	6	5	7	8	8	2
150–199	7	1	2	2	1	2	2	0	2	3	3	0
200–249	6	1	2	2	2	2	3	2	5	3	3	1
250–299	3	1	2	2	1	3	3	1	3	3	3	1
300–349	2	1	0	1	0	1	1	0	1	1	1	0
350–399	5	1	2	2	1	2	2	1	2	2	2	1
400 or more	5	3	3	3	3	4	4	3	4	4	4	3
Totals	54	11	15	19	12	20	21	12	24	24	24	8

[1] Number of hospitals in this column is reduced by failure in some instances to take action against division of fees.

PRACTICAL APPLICATION OF THE MINIMUM STANDARD

The practical application of the minimum standard to the hospital and the meaning of this standard, when in effective operation, to the community, are here considered.

The primary purpose of nearly all hospitals is the care of the sick or injured. This means that, as a matter of policy, the hospital seeks to render to each patient admitted the most efficient care known to the staff of the hospital. Hospitals and doctors accept this interpretation; otherwise the hospital would be merely a boarding-house for the sick or injured. Further, the trustees of the hospital, having accepted this policy, are responsible for the administration of the policy; and the people of the community have a right not only to assurance that the policy is carried out but

also to the facts upon which such assurance is based. It is only upon such a relationship of mutual confidence that the hospital may reasonably ask the good will and support of the community. Again, upon such a relationship rests the ultimate success of the hospital. The minimum standard is designed to foster just this fundamental relationship.

If the board of trustees is responsible that every patient, free or pay, in the hospital receives the best care known to the staff, then the board must at frequent intervals be in possession of the facts as to the care received by the patients in the hospital. The board must know, for example, if any unnecessary surgical operations are performed in the hospital; or if incompetent surgical operations are performed; or if lax, lazy, or incomplete diagnoses are made. If infections occur, the board must know, as nearly as may be, the cause of the infection, and it must know that every reasonable effort is made to remedy the cause. If the time of patients is wasted between their admission to the hospital and the proper study, diagnosis, and treatment of their illness, again the board must know the facts and take action promptly to prevent further waste of this kind. Too frequently hospital trustees consider that their duties end with the management of the financial affairs of the hospital.

Now let us suppose that in a given city the leading hospital has "put on a campaign" to raise $100,000, and that the chamber of commerce, desiring to help the hospital, sends its representative to the trustees to ask whether or not the service at the hospital is what it should be. In other words, the chamber of commerce wants facts; it wants the "production sheets" of the cure and relief of illness.

What can the trustees answer? If the hospital is one which meets the minimum standard, the trustees are in position to say: "We have the facts as to the clinical successes and failures in the hospital. We give you these facts and rest our claim for support on them."

But if the hospital is one which does not meet the minimum standard, the trustees will probably reply: "We hope that all of our patients receive the right sort of care. We believe that they do. But we really know very little about the matter." This answer is sentimental rather than businesslike and will seldom win today the confidence of an intelligent community.

In case of the latter reply the chamber of commerce, and in fact the entire community, will do well to withhold support until the hospital produces evidence that it is worth while. Among the 671 general hospitals of 100 or more beds in the United States and Canada, about 468 of them cannot present at this time even a fairly complete analysis of their clinical work.

Let us suppose now that the trustees of one of these 468 hospitals decide that they will seek in a businesslike way the confidence of their community. What is their procedure?

Can the trustees "inspect" the hospital and through this means make a report concerning the clinical work in the hospital? The answer is no.

What the trustees can do is to call the doctors together who practice in the hospital and put the matter squarely up to them. After a statement of their own responsibility in the matter, the trustees can say: "We want you to organize as a group or staff and to create among yourselves a group-consciousness (Minimum Standard, 1). You are the experts. We ask that you draw up rules and regulations which if wisely administered will, in your judgment, provide for each patient in the hospital the highest service which is in your power to give (Minimum Standard, 3). We ask, further, that you meet as a staff at least once each month and that at these meetings you analyze your work in the hospital; that you find your mistakes or failures and, in so far as you can, prevent recurrence of the same mistakes or failures (Minimum Standard, 3, *a* and *b*). We ask your co-operation, your guidance, and your confidence; we ask for the facts from time to time with regard to the clinical service in the hospital. In turn, we pledge our support."

The College in its work among hospitals has not found a single group of doctors practicing in a hospital who would not respond gladly to

such a request from the governing board of the hospital. Here and there certain "obstacles" to the plan are raised. The chief of these is that the doctors are too busy to hold regular meetings and analyze their work. But in answer let us ask: Is any doctor too busy to give his patients the best service in his power?

After the staff has organized and after it has agreed upon rules and regulations governing the professional work in the hospital, and after these have been approved by the trustees, then it becomes the obligation of the trustees, through the superintendent of the hospital, to administer or to carry out the rules and regulations. It is essential that a spirit of co-operation exist between the superintendent and the staff in the administration.

ANALYSIS OF CLINICAL RECORDS

In order to make more clear what is meant by an analysis of the clinical service of a hospital, let us consider the following two series of 100 operations for chronic appendicitis, both of which are fairly typical of what happens today in hospitals.

	In hospital No. 1	In hospital No. 2
Complete physical examination, including blood count	100	14
Number of consultations held	41	2
Working diagnosis recorded in advance of operation	100	None
Progress notes recorded by doctor	100	None
Incorrect diagnosis	1	14
Infections following operation	3	12
Number patients relieved	94	77
Number patients died	3	9

The analysis of the cases treated in hospital No. 1 shows that a complete physical examination was made and recorded for each patient; that in order to clear away doubt as to the diagnosis, consultations were held in 41 cases; that the working diagnosis in each case were, then, in fairness to the patients, recorded in the permanent records of the hospital; that, after the operations, the physicians or surgeons in charge of each case made or signed daily a statement of the progress of the patient; that infections developed in 3 cases; that 4 incorrect diagnoses were made; that the number of patients apparently relieved of their illness was 94; and that 2 of the patients died following operation. This record is a credit to the staff of the hospital.

The corresponding data are now given for a similar series of cases in hospital No. 2. The data as here presented could not occur in a hospital which meets the minimum standard. In a hospital which meets the minimum standard, for example, it is not possible that any patient, except in an emergency, will go to operation in advance of a complete physical examination. But in hospital No. 2 86 of the patients were operated upon without a complete physical examination. They were operated upon, it seems, after guess-diagnoses rather than after scientific diagnoses with consultations when indicated.

Considering the record of hospital No. 2, is there anything unreasonable in asking that the staff meet at least once each month, that it analyze the facts of its clinical work, that it determine, as nearly as may be, the causes of its failures; and that, demanding the support of the trustees, it endeavor to remove these causes? For example, 12 of the cases developed infection. Whose cases were these? What is the nature of the infection as indicated by laboratory analysis? Were the cases operated upon in rooms where pus cases had also recently been operated upon? Is the sterilization in connection with the operating room effective? When was it last tested and how? What technique is carried out in connection with surgical operations? And again, what were the reasons for the incorrect diagnoses? Do they indicate a lack of study, undue haste, or a failure to make full use of the laboratory facilities?

If the staff of hospital No. 2 would in dead earnest ask such questions as these each month, the percentage of infections would undoubtedly decrease. If the staff-review were really penetrating, the percentage of deaths would undoubtedly decrease. Matters, too, of incompetence, when the facts indicated incompetence, would be dealt with in no uncertain manner. The staff would become restricted (Minimum Standard, 2). The doctor scarcely exists who, if incom-

petent and if his incompetence is brought to light at frequent intervals, will not either endeavor promptly to perfect his training or retire from membership on the staff. The same principle is true with regard to character and professional ethics. Can any staff rest content with less than its maximum effort at all times to perfect the service of its hospital?

But the division of surgery, or the surgical cases, should not alone be analyzed by the staff. The clinical records of non-surgical cases merit quite as much attention. The study, diagnosis, treatment, and final result in pneumonia cases, cases of malnutrition, etc., should be reviewed. The services of the obstetrical department and of the children's department should, in the same way, be included.

In making analyses of clinical work, the confidential character of no record should be violated. The data for consideration of the staff may be taken from summary cards from which the names of patients are omitted. See Bulletin, Vol. IV, No. 1.

Various hospital staffs at this time are in process of finding out for themselves the most effective means of analyzing their clinical records. The data as indicated on page 20, Bulletin, Vol. IV, No. 4, are found by a number of hospitals to be of stimulating value when presented to the staffs. Copies of these data are placed in the hands of each member of the staff; point by point the data are reviewed and the responsibility for the character of the data is shared by each staff member.

In order to compile the data called for on the sheet, it is suggested that a daily review be made of the records of patients discharged. This review will include information under each of the headings of the sheet. When a daily record is kept in this fashion the summary for the month is merely a matter of arithmetic.

From time to time exhibits are made showing these data comparatively through a series of months. In addition to these data some definite report of the laboratory service of the hospital is helpful. This report should show the extent to which the laboratory is used; and in this connection questions as to the adequacy and competence of the laboratory service should be raised by the staff. The staff should especially recognize good work on the part of the pathologist. All gross material removed at operations should go to the pathologist for report; and the case record of each death in the hospital together with the autopsy record when available should be presented as a routine at the next succeeding staff meeting.

Explanation of the practical application of the minimum standard which grew out of the experience of the Staff at St. Vincent's Hospital, Los Angeles, and at The Woman's Hospital, New York, are given in this pamphlet. In this connection the staff rules and regulations as given in the appendix (Bulletin Vol. IV, No. 4) may also be helpful.

THE DIVISION OF FEES

In connection, further, with restricting the privilege of practice in hospitals, emphasis is placed upon the division of fees, or fee-splitting (Minimum Standard, 2). Fee-splitting is the buying and selling of patients. The practice exists in various forms, but the most usual form is as follows: A general practitioner makes a diagnosis in which surgical interference is indicated. He then refers the patient to a surgeon for operation. The surgeon operates, collects a fee, and sends to the physician one-third or one-half of the fee, this last transaction being unknown to the patient. Sometimes the physician collects the fee "for the surgeon" and retains his percentage as agreed with the surgeon.

Sometimes the fee is divided with the explanation to the patient that the physician "assists the surgeons" or gives the anæsthetic. In many such instances the explanation is a subterfuge for fee-splitting. A competent surgeon usually has a regular assistant and an anæsthetist with whom he is accustomed to work and is more able in this way to do good work than if he permits each referring doctor to assist him.

Undoubtedly the physician should be paid for the study and diagnosis of a surgical case. But he should be paid directly for this service by the patient. In the same way the surgeon should be paid directly by the patient. The

surgeon can frequently be of service to the physician and to the patient by explaining to the patient the value of the study and diagnosis made by the physician. But the accounts of the physician and of the surgeon should not be confused or rendered to the patient as a single statement.

The evils of fee-splitting are, first, that it makes for incompetent surgery. The surgeon who is party to the practice gets his cases usually not upon the basis of merit, but upon the basis of the percentage of fees collected that he will give to the practitioners. The more incompetent he is, as a rule, the larger percentage of the fees he gives to his co-fee-splitters.

Second, fee-splitting makes for unnecessary surgical operations. Under the fee-splitting system surgery becomes a commercial enterprise and not a professional service. Both the physician and the surgeon tend to make surgical diagnoses without adequate study, and the result is unnecessary surgery. Much of the unnecessary surgery of our present day is due directly to fee-splitting.

Third, fee-splitting, by introducing dishonesty into medical practice, lowers the entire medical profession in the estimate of the public. The fee-splitter, for example, says to his patient that he refers him to a most competent surgeon, when he knows well enough that if he, the physician, were to be operated upon, he would select another surgeon. Further, the fee-splitter usually poses before his patient as having received little or no fee for his services when, as a matter of fact, he has received a large fee indirectly from the patient. He holds such a fee really as a theft.

Fee-splitting is now prohibited by law in the following states: Minnesota, Wisconsin, Nebraska, West Virginia, Alabama, Ohio, Kansas, Iowa, Tennessee, Colorado, Michigan, and Georgia.

The great majority of physicians and surgeons are most sincerely eager to put an end to all fee-splitting. They ask the hospital trustees to help them in this matter by excluding fee-splitters from the privileges of practice in hospitals.

The College asks each hospital to make clear to its staff and to its community a definite policy against fee-splitting. Where no action has been placed on record in this matter, the College suggests that the governing boards of hospitals pass the following resolutions:

> *Be it resolved*, That the practice of the division of fees is inconsistent with the policy of ..... Hospital and that physicians and surgeons who divide fees are not permitted to practice in the Hospital; and further,
>
> *Be it resolved*, That a copy of these resolutions be sent to each physician and surgeon now practicing in the Hospital.